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Archives of Neurology and Psychiatry

VOLUME 13 APRIL, 1925 NUMBER 4

TUMORS OF THE PINEAL BODY*

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AND
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BOSTON

Tumors arising from the pineal body (the epiphysis cerebri) are among the rarer types of intracranial new growths. They are interesting from three points of view: first, from a purely neurologic standpoint, since they give rise to symptoms which have been of value in the localization of tumors in the pineal neighborhood; second, from the standpoint of the internal secretions, because of the much mooted question of a possible endocrine function of the organ; third, from a histopathologic standpoint, because of the peculiar and poorly understood structure of the epiphysis and consequently of the tumors which arise from it.

Numerous investigators have been inclined on an anatomic basis to believe in the glandular nature of the organ in question; and from their recent phylogenetic studies, Tilney and Warren [1] have argued for such a belief. Physiologic experiments with pineal extract, however, have given negative results. Extirpation of the pineal body in animals has been rather unsatisfactory on the whole, and evidence of function from this source has at the best been inconclusive. The same can be said of feeding experiments. Several noteworthy reviews of all previous work relating to the epiphysis have appeared from time to time, the most recent and complete being that of Krabbe.[2]

It becomes evident, then, that it is from clinical observations on verified cases of pineal tumors that our most dependable information can be secured, not only as to neurologic data, but also in respect to any possible function of the organ. Such function, whether actual or

*From the Surgical Clinic of Dr. Harvey Cushing, Peter Bent Brigham Hospital, Boston.

*Read at the Fiftieth Annual Meeting of the American Neurological Association, Philadelphia, June, 1924.

1. Tilney and Warren: The Morphology and Evolutionary Significance of the Pineal Body, Am. Anat. Memoirs, Philadelphia **9**:7-257, 1919.

2. Krabbe: The Pineal Gland, Especially in Relation to the Problem of Its Supposed Significance in Sexual Development, Endocrinology **7**:379-414 (May) 1923.

assumed, concerns changes in primary and secondary characteristics of sex which take place before the age of puberty, the so-called syndrome "macrogenitosomia praecox" (Pellezzi) or "pubertas praecox." Thus it is only cases of pineal tumor occurring in childhood from which this particular information can be derived. General neurologic and localizing data, however, may be obtained from cases of pineal tumors in patients of all ages.

The object of the present communication is to report, from a combined clinical and histopathologic standpoint, the twelve verified pineal tumors in Dr. Cushing's series, three of them from his Johns Hopkins' records and nine from those at the Peter Bent Brigham Hospital. To elucidate their clinical aspects, these cases had been originally divided on clinical grounds into: (1) the five cases occurring before the age of puberty, and (2) the seven cases occurring in adult or at least postpubertal life. However, since there appears to be no definite correlation between the histologic character of the lesion and the occurrence of Pellezzi's syndrome, it has seemed best to us to present both the prepubertal and postpubertal cases in the chronologic order in which they appear in the records, and to reserve for the end a general discussion of the neurologic, endocrinologic and histopathologic features of the series of cases as a whole.

As a description of the neoplasm will be appended to each of the case histories, it is perhaps advisable at this point not only to describe briefly the histologic methods which have been employed in their study, but also to explain the terminology which has been used. Although many designations have in the past been given to pineal, as to other tumors, they appear to us to be of two essential varieties. The more common tumor is what Krabbe has termed a *pinealoma,* in other words, a tumor composed of elements of the normal epiphysis; and of these we would call attention to the fact that two forms are recognizable: (1) a spongioblastic form representing the early undifferentiated cells of the organ, and (2) a form which is composed of pineal cells of adult type. The less common variety of tumor is a *teratoma,* a type of growth unmistakable under the microscope, of which only one example occurs in this series of twelve cases.

HISTOLOGIC PROCEDURES

In studying these cases, we have employed the special methods for displaying the several component structures of the pineal body. All the tumors were fixed in 10 per cent. liquor formaldehydi, and the following staining methods were used: *on frozen sections,* (1) Bielschowsky's, (2) Hortega's first and fourth variants of Achúcarro's tannic silver method, (3) Perdrau's and (4) Hortega's method for pineal paren-

chyma; *on paraffin sections,* (1) Heidenhain's iron hematoxylin, (2) neutral ethyl violet-orange G (Bailey), (3) phosphotungstic acid-hematoxylin (Mallory), (4) hematoxylin and eosin, (5) Perdrau's modification of Bielschowsky's method and (6) Walter's gold chlorid method.

Hortega's method for pineal parenchyma is as follows; the others are well known and easily available.

1. Fix in 10 per cent. liquor formaldehydi for at least two days.
2. Make frozen sections as thin as possible.
3. Treat for twenty-four hours at room temperature in the following solution:

   Ten per cent. silver nitrate solution, 10 c.c.
   Pyridin (pure), 3 drops.

   The sections become dark yellow.
4. Wash in distilled water to which have been added 2 drops of pyridin.
5. Color in the following solution, heating to about 50 C., until the sections become a dark sepia color:

   Solution of silver carbonate, 10 c.c.
   Pyridin (pure), 3 drops.
6. Wash in distilled water.
7. Reduce in 10: 100 liquor formaldehydi.
8. Tone with gold chlorid, warming slightly to intensify the color.
9. Fix in 5 per cent. hyposulphite of soda.
10. Wash, dehydrate, clear. Mount.

For photographic purposes we have always used Walter preparations, in preference to those by Hortega's method, because, being so much thinner, better photographs may be obtained from them.

DEVELOPMENT AND COMPOSITION OF THE CORPUS PINEALE OF MAN

The following account of the early appearance of the epiphysis is taken from Krabbe:[3]

The pineal body is first evident in the second fetal month. Its anlage consists of two parts, a cell mass, and a fold posterior to it, in the roof of the diencephalon (Fig. 1). In the course of development, the cell mass enlarges and the fold becomes deeper; at the same time, its walls thicken. Throughout fetal life, the two anlagen are separated by a connective tissue septum. The cavity of the fold—the diverticulum pineale—is closed from the sixth fetal month onward.

In the first six months of fetal life, the cells, out of which the two pineal anlagen are composed, are small and round, with scanty protoplasm; only the walls of the diverticulum show cylindrical cells with cuticle and cilia. The cells continue to multiply by mitotic division throughout fetal life.

In the sixth fetal month, the rounded cells begin to differentiate. They develop more protoplasm and larger nuclei, which are, however, poorer in

3. Krabbe: Histologische und embryologische Untersuchungen über die Zirbeldrüse des Menschen, Anat. Hefte, No. 1, Pt. 163, p. 192.

chromatin. This metamorphosis of the cells does not take place all over the organ at the same time, but begins in spots and spreads from them. This phase of the metamorphosis lasts to the end of the first extra-uterine year.

The further development from the end of the first year to the adult condition is not well known. We will remark only that the connective tissue seems to come from the vessels and not from the capsule. We pass, therefore, directly to the adult organ, which, according to

Fig. 1.—The pineal anlage of man at the fifth fetal month (from Krabbe) to show the cell masses which develop from the two portions of the structure; also the type of cells of the primitive spongioblasts (Cf. Fig. 16).

textbook descriptions, is said to be made up of three types of cells: (1) what appear to be nerve cells provided with tuberous appendixes and processes ending in bulbs; (2) pineal cells proper or specific parenchymatous cells without appendixes and possessing some structural characteristics interpreted as secretory, and (3) cells with fibrillary expansions, apparently neuroglia.

The recent investigations of Hortega,[4] however, have shown conclusively that the *lobules of the pineal body are composed solely of two categories of cells, the parenchymatous cells and the neuroglia cells.* In effect, the supposed nerve cells and the specific cells (Types 1 and 2 above) are merely two aspects of a single parenchymatous cell type, and when they are properly impregnated it may be seen that both, at least when they have reached a certain stage of development, are provided with expansions and long processes ending in bulbs (Figs. 2

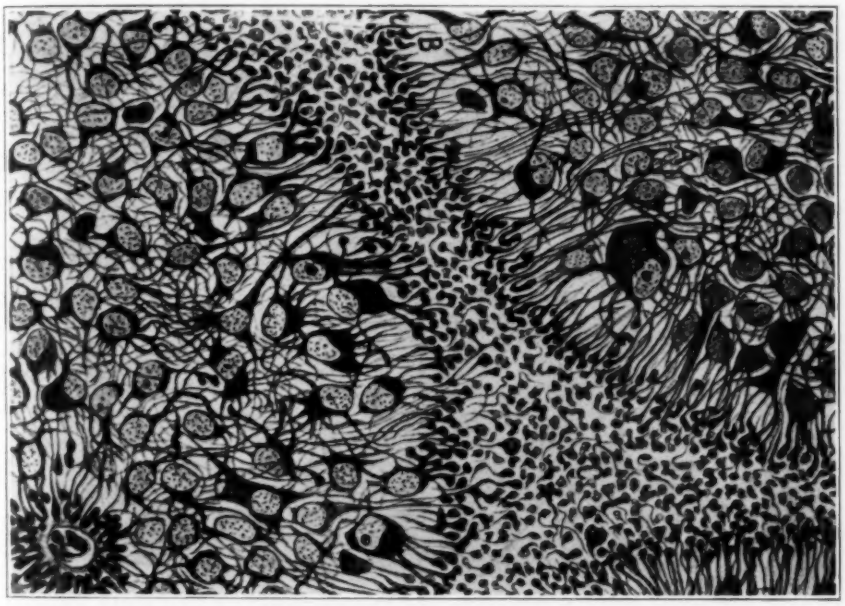

Fig. 2.—Microscopic drawing of the normal pineal body of a young adult (from Hortega), showing pineal parenchyma cells with their end bulbs; neuroglia unstained.

and 3). When impregnated in this way, however, it will be found that some of the parenchymatous cells are stained much more heavily than others (Fig. 3). These heavily stained cells, which are sometimes impregnated by the methods of Bielschowsky, Cajal or Golgi, were formerly and erroneously interpreted as nerve cells, although it was always recognized that they had neither Nissl bodies, neurofibrillae nor axons.

Concerning the nature of these parenchymatous cells, we can only conclude that they constitute a proper category of cells, being neither

4. Hortega: Constitucion histologica de la glandula pineal, Arch. de Neurobiol. **3**:359, 1922.

neuroglia, nerve cells, nor ependymal cells. They stand much closer to ependymal cells than either to neuroglia or nerve cells. They possess in their cytoplasm typical blepharoplasts like the ependymal cells. They remain for a much longer period in their development devoid of processes than do either the neuroglia or nerve cells. The development and complexity of their cell processes increase throughout life. In addition to these parenchymatous cells, which are the chief element of the pineal body, typical neuroglia cells also occur, but they are comparatively rare and constitute a minor feature of the structure.

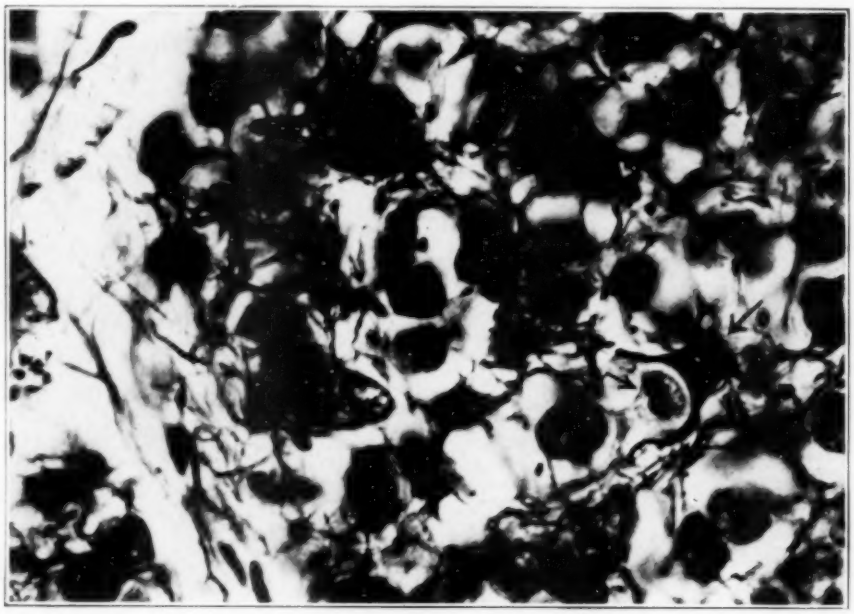

Fig. 3.—Parenchyma from normal pineal body of a boy of 17 (Hortega's method; X 850), to show the two types of parenchyma cells (cf. arrows), the one hardly impregnated and the other heavily impregnated. Since the section is very thick, the entire extent of the cell processes is not shown. One typical end bulb is seen in the upper left hand corner.

REPORT OF CASES

The first case in the series, observed just twenty years ago, was reported in another connection in a paper by Dr. Cushing.[5] It was stated therein that the tumor was "a glio-sarcoma of rather unusual character," a not uncommon diagnosis for tumors originating in the pineal body in days gone by.

5. The Establishment of Cerebral Hernia as a Decompressive Measure for Inaccessible Brain Tumors, Surg., Gynec. & Obst. **1**:305 (Oct.) 1905.

CASE 1.—*Pinealoma of spongioblastic type producing advanced signs of tumor in the contiguous area and internal hydrocephalus in a man.*

History.—Roy L., an engineer, aged 28, admitted to the Johns Hopkins Hospital on Oct. 23, 1904, was referred by Dr. Frank R. Smith with a diagnosis of brain tumor. His symptoms, attributed to the shock from dynamite explosions at the time of the Baltimore fire, were chiefly headache, vomiting and loss of weight. A neurologic examination showed little else than a high grade of choked disk and an unsteady gait with a tendency to fall backward. There were no cranial nerve palsies.

After a brief and ineffectual antisyphilitic regimen, a bilateral subtemporal decompression was performed, followed by improvement in his general condition

Fig. 4 (Case 1).—Pinealoma in median section. Note the widely dilated third ventricle, the peculiar trapezoid form of the growth, and the degree of compression of the cerebellum.

but by secondary optic atrophy and loss of vision. He was discharged, and reentered the hospital some months later.

The brief history states that "he gradually lost his hearing, became stone deaf and finally for the terminal three months lapsed ihto a passive state like a decerebrate animal whose bulbospinal axis alone remained active." The blindness and deafness, both of which were absolute, were supposed at the time to be associated with his general condition rather than indicative of the seat of the lesion. He died, June 15, 1905, of inhalation pneumonia.

Necropsy Findings.—The brain was removed after fixation *in situ* and showed (Fig. 4) "a tumor the size of a hen's egg which embraced the region of the corpora quadrigemina and had invaded equally on each side the region of the geniculate bodies, thus accounting for his deafness." It had obstructed the aque-

duct, and there was marked ventricular dilatation, particularly of the third ventricle. Attention may be drawn to the outline of the quadrilateral shaped tumor as shown by the mesial section and the close resemblance it bears to one or two other cases in the series (compare Figs. 7 and 33).

Reexamination of the tissue showed the tumor to be composed of broad pear-shaped cells (Fig. 5). Many of them were attached by their smaller extremities to the walls of blood vessels. The nuclei varied greatly in size but were usually large and ellipitical. Mitoses were rare. The centrosome was almost invariably a single heavy granule, lying in the cytoplasm near the nucleus; occasionally a diplosome was seen. In places, the cells were divided into pseudo-acini by an

Figure 5

Figure 6

Fig. 5 (Case 1).—General appearance of the tumor. Phosphotungstic acid hematoxylin stain; X 80. Note the pear-shaped cells with tendency to collect around the blood vessels.

Fig. 6 (Case 1).—Processes of the embryonic cells (spongioblasts). These are not neuroglia fibrillae and are not stained by phosphotungstic acid hematoxylin. Hortega's fourth variant; X 850.

abundant connective tissue stroma. Adult neuroglia cells and adult pineal parenchyma cells could not be identified. Throughout most of its circumference, the tumor was sharply demarcated from the surrounding brain tissue. The tumor in brief was composed of unipolar spongioblasts such as are frequently found in tumors of the cerebral hemispheres (Fig. 6).

This, then, was an example of a pineal tumor in an adult, which probably would have been recognized at least as a tumor of the corpora quadrigemina at the present day. The patient almost certainly must

have shown paralyses of conjugate movements, but these were not recorded, and, if observed, may in all likelihood have been ascribed to his blindness. The case was studied and reported rather from an operative point of view than from a neurologic or pathologic one.

Like the foregoing, the next case showed an advanced lesion with involvement of the corpora quadrigemina and complete deafness before the end. A regional diagnosis was fairly unmistakable, but there was no suspicion at the time that the lesion was of pineal origin.

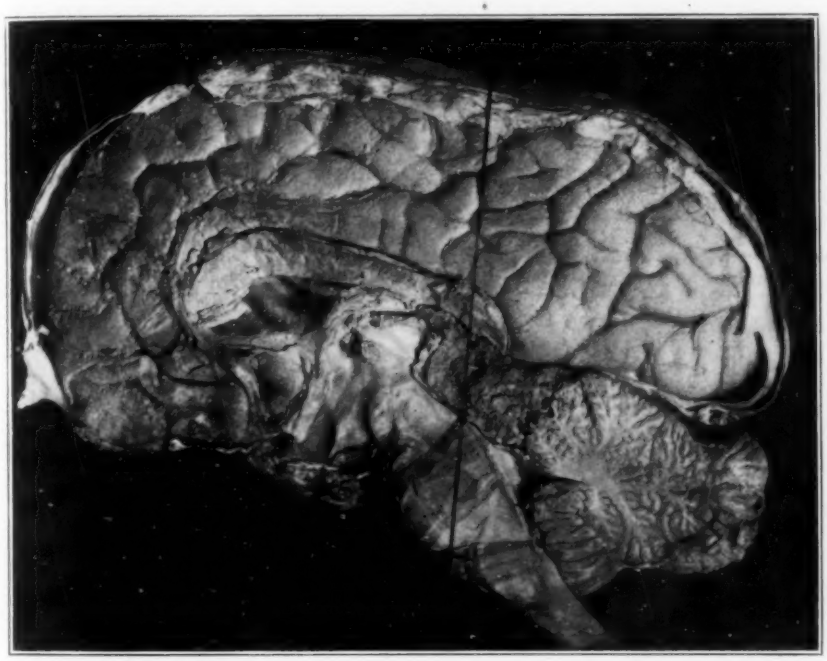

Fig. 7 (Case 2).—Tumor on midsagittal aspect and line of coronal section passing through nodule of surface tumor at pontomedullary junction.

CASE 2.—*Pinealoma of spongioblastic type causing advanced symptoms pointing to the area contiguous to the corpora quadrigemina with hydrocephalus in a woman.*

History.—Mrs. D., aged 29, admitted to the Church Home and Infirmary, Baltimore, on Oct. 9, 1907, had a negative family and past history. Her present illness, of about fifteen months' standing, began with headaches which became progressively more frequent and severe. After six months, her vision commenced to fail, and she had been totally blind for two months before entrance. Diplopia had been present before vision was lost. She also had complained of "noises" in her head for several months prior to admission, and hearing was gradually lost in both ears during this time. For two months, she had had generalized convulsive seizures.

Physical examination was almost exclusively objective, as she was blind, deaf and incooperative. The fundi showed choked disks with complete secondary atrophy. There was a right facial weakness and indefinite oculomotor palsies with lack of parallelism of the eyeballs. She had a positive Romberg sign, and there was marked suboccipital tenderness.

It was assumed that there was a posterior lesion, and on Oct. 14, 1907, the cerebellar region was explored, with negative findings and an early fatality. Permission for an examination, restricted to the head, was secured.

Necropsy Findings.—Sagittal section of the brain (Fig. 7) showed a tumor mass about 2.5 cm. in diameter, occupying the position of the pineal body, no

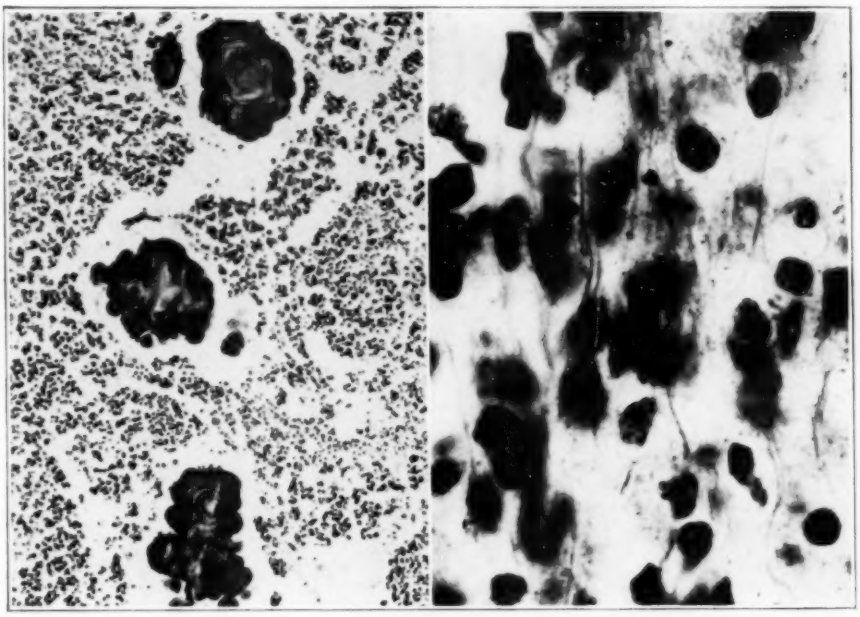

Figure 8 Figure 9

Fig. 8 (Case 2).—Calcium deposits. Phosphotungstic acid hematoxylin stain; X 80.

Fig. 9 (Case 2).—Processes of spongioblasts. Hortega's fourth variant; X 850.

trace of which was to be seen. The tumor had flattened the nervous tissue above the aqueduct of Sylvius into a thin band. The ependyma of the aqueduct could be followed all the way from the third to the fourth ventricle. The tumor infiltrated the meninges of the anterior surface of the cerebellum, and this infiltration had entirely encircled the brain stem, as may be seen from a celloidin transverse section of the entire left hemisphere, cut in the plane indicated in Figure 7.

On histologic study, the tumor was found to be composed of a connective tissue vascular matrix, supporting masses of small round or oval cells. These cells had a round, deeply staining nucleus and scanty cytoplasm. Delicate processes of the cells occurred in moderate numbers (Fig. 9). The centrosomes

of these cells consist usually of a single granule; occasionally a diplosome is seen. Here and there occur deposits of calcium (Fig. 8). The tumor in brief is composed of embryonic cells of the pineal anlage, tending to differentiate into neuroglia. It may properly be designated as a spongioblastoma.

In this case, as in Case 1, both the location of the lesion and the comparative futility of any operative procedure would certainly have been clearly appreciated today.

The following history records the first of the prepubertal cases in the series. The child showed a slight increase in stature in excess for his age, but this could hardly have been regarded as an evidence of Pellezzi's syndrome.

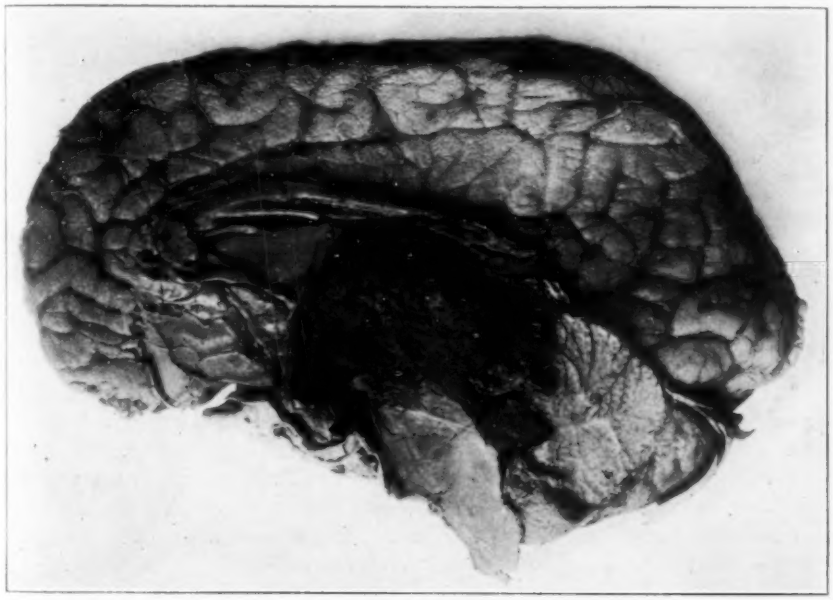

Fig. 10 (Case 3).—Massive interstitial hemorrhage in tumor and its relation to neighboring structures. The fourth ventricle is entirely filled and the cerebellum pressed on.

CASE 3.—*Pinealoma of spongioblastic type in a boy with atypical signs in the contiguous area and hydrocephalus in the absence of pubertas praecox.*

History.—Charles B. C., a boy, aged 6, referred by Dr. I. P. Lyon of Buffalo, N. Y., was admitted to the hospital on Feb. 8, 1912, with the complaint of headaches, vomiting and dizziness. He was said to have "developed exceptionally well." He showed no abnormality until six months before he came to the hospital, when a general lassitude was noticed by his parents, but he continued in school until two months before admission. At that time he began to have frontal headaches, with vomiting, and there was gradual and continual failure of vision. One month before admission, he had marked stiffness of the neck with a positive Kernig's sign, and several lumbar punctures were performed, but the

fluid showed nothing abnormal. Since then there had been marked loss of weight, extreme nervousness and irritability.

Physical examination revealed a very large boy for his age, his height being 128 cm. (normal for 6 years, 112 cm.). The circumference of his head was 58 cm. (normal for 6 years, 52 cm.). There was marked stiffness and soreness of the muscles of his neck, and cracked-pot resonance on percussion of the skull. The left eye was practically blind, and vision was greatly reduced on the right. The fields showed general contraction. There was a choked disk of 2 to 3 diopters on the right side, with much new tissue formation, and on the left a nearly complete secondary atrophy. The left pupil was larger than the right, but both reacted to light and in accommodation. There was a left abducens palsy. Kernig's sign was positive, and neither the knee nor the ankle reflexes could be elicited on either side. No pubic, axillary or facial hair was present. The genitalia were of normal appearance for his age.

On February 11, the lateral ventricle was punctured, disclosing hydrocephalus. On the presumption that there was a cerebellar tumor, the next day a suboccipital exploration was performed without disclosing the lesion and with a fatality four hours later. Necropsy was obtained, restricted to the head; postmortem time was not recorded.

Necropsy Findings.—Sagittal section of the brain (Fig. 10) showed a dark red central tumor mass extending into the third and fourth ventricles, to the naked eye resembling a large intraventricular hemorrhage. The mass was attached to the anterior surface of the cerebellum. No trace of the pineal body could be found. It will be noted that the splenium of the corpus callosum is flattened, and that the distance between it and the brain stem is disproportionately great.

Microscopic examination showed the tissue to be poorly preserved. The tumor mass is composed of an intimate mixture of red blood cells and tumor cells. Evidently, a hemorrhagic softening had occurred in the tumor. The only place in which the relatively normal structure remained was along the anterior surface of the cerebellum. Here the tumor may be seen to infiltrate the meninges over the cerebellar cortex (Fig. 11), even invading the cortex at places along the perivascular spaces. It is everywhere definitely outside the nervous tissue.

The tumor appears to be composed of embryonic neuroglia cells (spongioblasts). They have large vesicular nuclei and very little cytoplasm. A few neuroglia fibrillae are seen (Fig. 12). Blood pigment abounds everywhere.

The one point indicating any possible endocrine disorder, as it is understood today, was the bodily overgrowth, but the mere increase in stature is of meager significance. There was nothing, either on a neurologic or somatic basis, to lead to the suspicion, twelve years ago at least, of a pineal tumor. It may be assumed that the ventricular puncture with its abrupt lowering of intracranial tension was what led to the extensive interstitial hemorrhage found in the tissue postmortem.

The following case,[6] which has been reported previously, was the first of the verified pineal tumors in the Brigham Hospital series. The

6. Horrax, G.: Studies on the Pineal Gland. II. Clinical Observations, Arch. Int. Med. **17**:627-645, 1916.

lesion was designated at that time as a pineal "struma," a noncommital diagnosis presumably intended to mean an adenomatous lesion. In view of our recent detailed histologic studies by more modern methods of staining, the case in abstract may deserve this second report.

CASE 4.—*Pinealoma of adult type with metastasis in third ventricle; xanthochromia; pubertas praecox; internal hydrocephalus; diagnosis of meningitis.*

History.—On Dec. 22, 1913, Samuel K. B., aged 12, was admitted to the hospital, a healthy, athletic boy who had grown rapidly, with an increase of 40 pounds (18 kg.) in weight in a year. At 11 years of age, he had acquired full

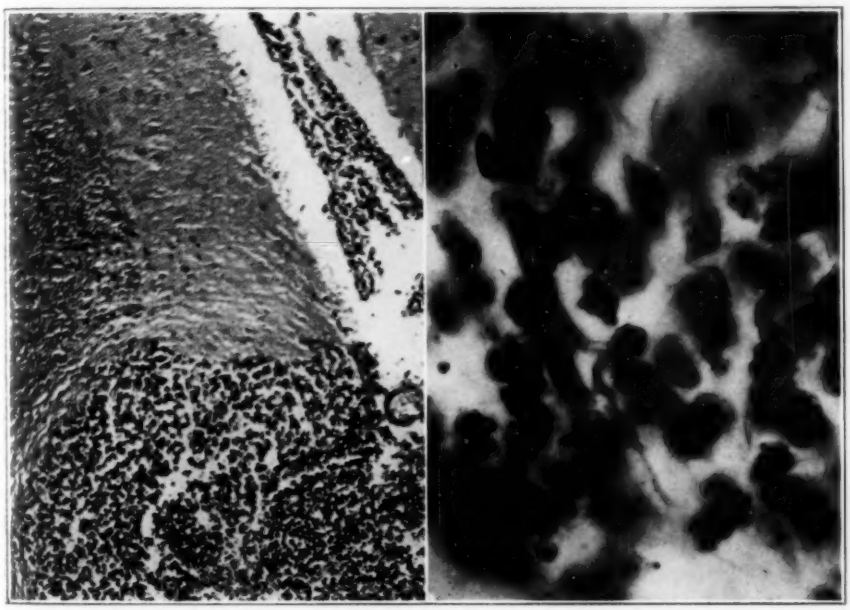

Figure 11 Figure 12

Fig. 11 (Case 3).—Tumor outside cerebellar cortex, infiltrating meninges. Hematoxylin and eosin stain; X 80.

Fig. 12 (Case 3).—Neuroglia fibrillae. Hortega's first variant; X 850.

secondary sex characteristics, with an adult voice. Accompanying this physical and sexual precocity, there was also a corresponding intellectual maturity in advance of his age. For three months before admission, there had been headaches, vomiting and temporary polyuria, and just before admission, diplopia.

Physical Examination.—This revealed a boy, 5 feet 3 inches tall, large and muscular for his age, with abundant hirsutes and beginning beard of adolescence. He showed slight oculomotor palsies, with diplopia, choked disk of 2 diopters and a positive Romberg sign, staggering gait and some ataxia. Lumbar puncture showed xanthochromic fluid. A decompression was advised and refused. He subsequently had some numbness of his right hand with a convulsion, and was readmitted to the hospital on Jan. 28, 1914, in coma, with a choked disk of 5 to 6

diopters. There was rigidity of the neck, and a positive Kernig sign, and a possible tuberculous meningitis with hydrocephalus was suspected. A ventricular puncture was performed, disclosing hydrocephalus with xanthochromic fluid containing a large number of old crenated red blood cells. There was temporary improvement followed by a return of coma and death.

Necropsy Findings.—Examination was made five hours after death and was limited to the brain. On bisecting the hemispheres, a tumor (Fig. 13) measuring 2.5 cm. in diameter was disclosed in the pineal region. In the center of the tumor was a large blood clot, and the lesion, as in the preceding case, was largely composed of a soft material full of interstitial hemorrhage. The ventricles were all greatly dilated, and in the floor of the third ventricle was a tumor implantation of considerable size.

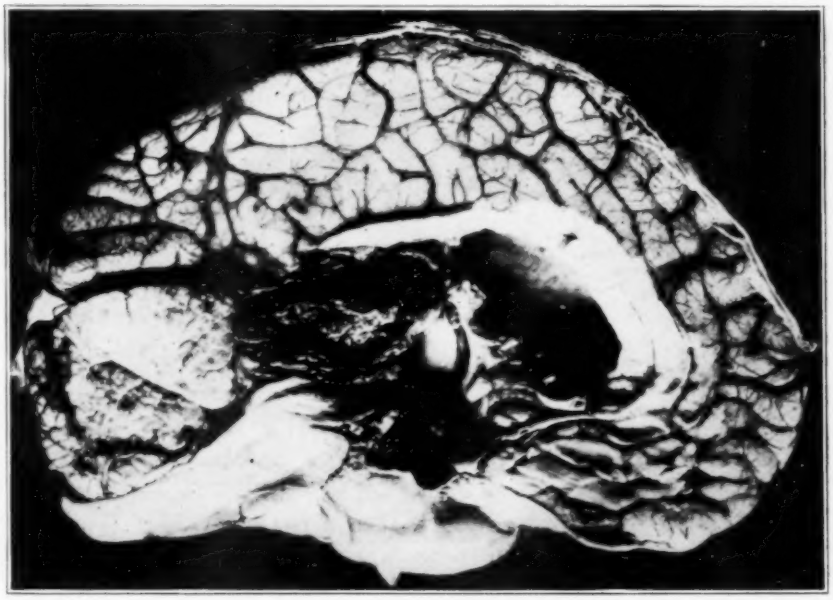

Fig. 13. (Case 4).—Note the compression of the posterior part of the corpus collosum and the implantation growth in the infundibulum.

The microscopic examination, in accordance with the earlier report, showed "a mass of loosely placed cells of epithelial character without any definite arrangement and no interstitial stroma, many of the cells being of the lymphoid type." The appearance was thought to resemble the cellular tumors (adenomas) of the hypophysis, then loosely designated as struma. There were many mitotic figures.

Reexamination of the tissue showed the supposed epithelial cells to be pineal parenchyma cells (Fig. 14). The lymphoid cells lie in the meshes of a stroma of reticular connective tissue. No neuroglia cells can be identified. The center of the tumor consists of a mass of pineal parenchyma cells of adult type, almost without stroma, with many areas of degeneration. The tumor is sharply circumscribed from the surrounding tissues. The previous diagnosis of "struma" of the pineal body we shall simply change to pinealoma (Krabbe) to coincide with our present terminology. We were unable to make special stains because the tissue

had been used in previous studies. Comparison with the following cases will enable one easily to interpret the appearances seen in Figure 14.

This case, then, shows a pinealoma which had been accompanied by unmistakable evidence of a precocious development in sexual, somatic and mental spheres, and it is surprising, in reviewing the case today, that at the time of his admission there should have been any doubt as to the diagnosis. This may possibly be explained by the fact that the patient's brother had succumbed two years before to a meningeal infection, which perhaps served to color the impressions of those who had the case in charge.

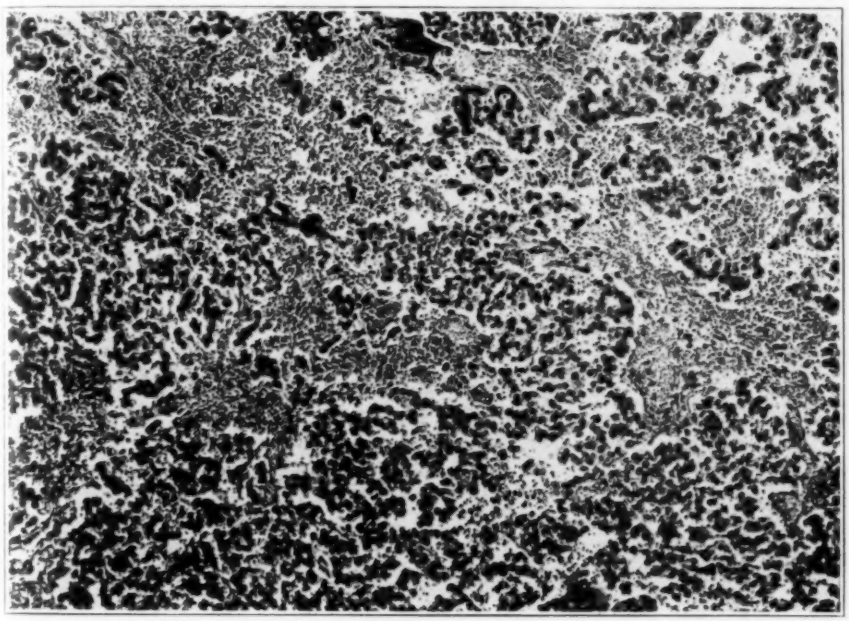

Fig. 14 (Case 4).—Note the large pineal parenchyma cells and the lymphoid cells of the connective tissue reticulum. Hematoxylin and eosin stain; X 80.

The following history is that of another prepubertal case, but as the patient was a girl, the macrogenitosomia praecox question does not enter, for in the female this syndrome, at least in any outspoken and recognizable form, does not occur. To be sure, cases of precocity are known in the female, but they are apparently confined to an association with suprarenal tumors.

CASE 5.—*Pinealoma of spongioblastic type with marked contiguous and general pressure symptoms, but no evidence of precocity.*

History.—Annie G., a girl, aged 10, referred by Dr. J. T. Moore of Houston, Tex., was admitted to the hospital on Oct. 23, 1916. Her family and past history

were negative. For a year and a half, this child had had frontal headaches. After a vacation from school, they ceased for several months, but about five months before her admission they recurred severely, and were accompanied with projectile vomiting. At the same time, vision began to fail, and on May 18, 1916, a decompression was performed. After this procedure, her vision and other pressure symptoms were relieved for a month or two, but by August, 1916, she became worse, acquired a staggering gait, and began to get deaf, first in the right ear and then in the left. Two lumbar punctures without relief were performed before her admission.

Physical Examination.—This showed a well developed and well nourished child weighing 70 pounds (32 kg.), slightly above the average for her age and sex. She was semistuporous and could scarcely be aroused. Cracked-pot resonance could be elicited on percussion of the skull, and there was tenderness over both suboccipital regions. Both pupils were widely dilated and did not react to light or in accommodation. There was a bilateral abducens paralysis and a choked disk of 5 diopters with much new tissue formation and advanced secondary atrophy. The right ear was completely deaf, and only very loud voice sounds were appreciated with the left. The deep tendon reflexes were not obtained on either side, but Babinski's sign was positive both on the right and on the left.

Treatment and Course.—On Oct. 27, 1916, a ventricular puncture was performed, 60 c.c. of clear fluid under increased pressure being withdrawn. After this procedure, the child was so far improved as to permit of a thorough neurologic examination. In view of the ataxia, a tentative diagnosis of cerebellar tumor was made. On Oct. 31, 1916, Dr. Cushing explored the cerebellar region, with negative results. A thick-walled posterior cistern was disclosed and a large amount of fluid evacuated. After this procedure, the child declined steadily, with a rising temperature which reached the extraordinary level of 110 F. just before her death on November 9. A necropsy, restricted to the head, was performed seven hours postmortem.

Description of Tumor.—The general relationships of the tumor found at necropsy may be seen in the photograph (Fig. 15). The anterior extent of the growth is not shown, since the brain was cut first in coronal sections, and the anterior portion was subsequently destroyed or misplaced. The note of the pathologist says that two large, soft, semigelatinous tags extended forward into the third ventricle, one downward into the infundibulum, the other projecting through the foramen of Monro into the right lateral ventricle.

The splenium of the corpus callosum was pushed upward and flattened. No trace of pineal body was seen. The tumor shelled readily out of its bed, its only attachment being to the meninges at the posterior extremity of the corpus callosum.

The microscopic structure may be seen in Figure 16. The tumor was composed of a mass of cells, with no definite architectural arrangement. Small, thin-walled blood vessels ran here and there. There was no other connective tissue. The cells were rounded or pear-shaped. No neuroglia fibrillae were found. The nuclei were round or slightly oval. No granules of any kind were to be seen in the cytoplasm of the cells.

In its histologic appearance, the tumor closely resembled the spongioblastic anlage of the pineal body (Fig. 1).

The following history records the only example of a teratoma in the series:

CASE 6.—*Teratoma pinealis with early precocity as well as marked signs of tumor in the contiguous area.*

History.—Russell B. P., aged 3, referred by Dr. K. G. Percy of Brookline, Mass., was admitted on Dec. 19, 1916, with the complaint of headaches, failing vision, vomiting and convulsions. The family history was negative. The child's

Fig. 15 (Case 5).—Note compression of cerebellum, corpora quadrigemina and corpus callosum. For relationships of anterior part of tumor see text.

past history was unusual. His first two teeth appeared at the age of 2 months, and he had seven teeth by the age of 4 months. He was talking at 8 months of age, and walked at the age of 9 months. When 5 months old, he weighed 25 pounds, or 11 kg. (normal for 6 months, 16 pounds, or 7 kg.). The mother said that he had always been extremely intelligent and far advanced for his age. He had learned to ride a velocipede and had been roller skating during this, his third, year. The present illness began acutely two or three days before admission with the onset of headache, followed by drowsiness and convulsions. A lumbar puncture, at the child's home, relieved these symptoms temporarily, and he was brought into the hospital the day after this puncture.

Physical Examination.—The child was 41½ inches in height (normal for age, 35 inches). He weighed 36 pounds, or 16 kg. (normal for age 31.2 pounds, or 14 kg.; Fig. 17). Conjugate movements of the eyeballs were impossible upward above the horizontal plane, and there was a right abducens palsy. There was a slight nystagmus to the right and left. The child's gait was rather ataxic and on a wide base. There was some slight ataxia of the hands in the finger to nose tests. His eyegrounds showed bilateral choked disks of 2 diopters. He was drowsy most of the time in the hospital, but was aroused easily, and he answered questions more intelligently than could be expected of a child of 3. A roentgenogram of the skull showed the marked convolutional atrophy of an internal hydrocephalus.

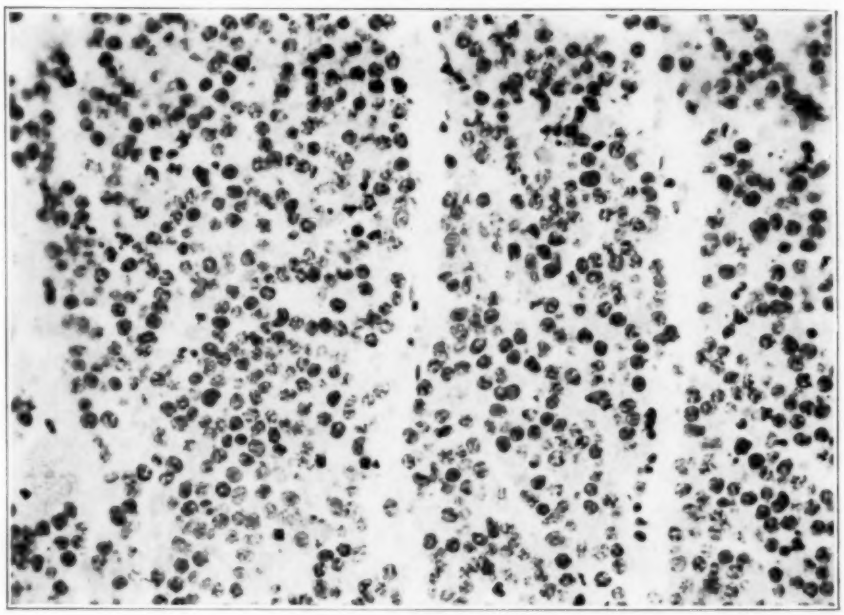

Fig. 16 (Case 5).—Tumor. Neutral ethyl violet-orange G stain; X 300. Note resemblance to pineal anlage, Fig. 1.

Treatment and Course.—The child was discharged on Jan. 25, 1917, without any operative intervention. During the next two years, Dr. Cushing was in the service in France, and nothing was heard of the case until March 5, 1920, when it was learned that the child had died at a neighboring hospital following a ventricular puncture.

Necropsy Findings.—Complete necropsy was obtained, time postmortem not known. This disclosed a typical cystic teratoma of the pineal body (Fig. 18), containing bone, cartilage, fat and hair. Microscopic examination of the tumor showed also epithelium resembling skin with a well marked horny layer, hair follicles and sebaceous glands (Fig. 19). There was marked dilatation of the ventricles. The testicles were reported as normal for his age, as were also the other internal organs.

This child showed an unquestioned physical and mental precocity, and though the notes regarding his sexual development are meager, the photographs (Fig. 17) would at least indicate that the genitalia were unduly large for his age. Bodily overgrowth and intellectual maturity were, however, two of the factors mentioned by Frankl-Hochwart [7] in his classical description of the syndrome of pineal tumor in children, and taken alone even without evidence at this early age of sexual precocity would be enough to aline this patient with the pubertas praecox cases.

Fig. 17 (Case 6).—Russell B. P., 3 years of age and 6½ in. above the normal height.

This had been the fourth successive prepubertal case and five years passed before another pineal tumor, this time in an adult patient, appeared in the clinic. Unless we have overlooked some cases which may have been incorrectly diagnosed and recorded, fourteen years had elapsed since the last adult (Case 2) had been studied. In the following, the next case in the series, the significance of the bilateral deafness was again disregarded at the bedside. The case was diagnosed as an acoustic (possibly bilateral) tumor.

7. Frankl-Hochwart: Ueber Diagnose der Zirbeldrüsentumoren, Deutsch. Ztschr. f. Nervenk. **20**:455, 1909.

CASE 7.—*Pinealoma of adult type with marked symptoms in the contiguous area and hydrocephalus.*

History.—On Dec. 5, 1921, Marcel T., a man, aged 23, entered the hospital, referred by Dr. S. C. Fuller of Boston, with a supposed cerebellar tumor. The family and past history were unimportant. It was stated that the patient had shown some mental changes for three years past. For one year, he had had blurring of vision and for ten months tinnitus in the right ear followed by impairment of hearing. For six weeks, he had complained of frontal headaches, and for two weeks of numbness on the right side of the body.

Physical Examination.—This showed bilateral choked disks of 5 to 6 diopters, weakness of the abducens on either side, hypesthesia of the right side of the face, arm and leg, as well as a right facial weakness of the central type. There

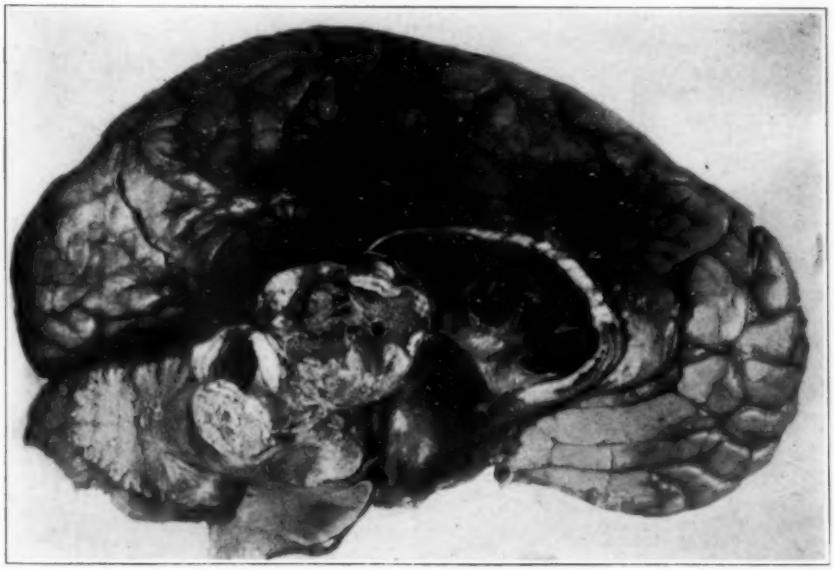

Fig. 18 (Case 6).—Typical teratoma. Note compression of cerebellum, corpora quadrigemina and corpus callosum, and enormous dilatation of the ventricular system.

was impairment of hearing in the left ear, and the right ear was completely deaf. In addition, there were marked signs of cerebellar involvement, including persistent nystagmus to the right and left, ataxia and incoordination of both upper and lower extremities, a positive Romberg sign and unsteady gait.

Treatment and Course.—In view of these findings, Dr. Cushing explored the cerebellar region on Dec. 17, 1921, but disclosed no tumor. The patient died four days later, and a postmortem examination was made, restricted to the head.

Necropsy Findings.—The brain, cut serially in coronal sections, contained an egg-shaped tumor mass, 3 cm. in length by 2 cm. in width, occupying the position of the pineal body. The growth was sharply delimited. The corpora quadrigemina were pushed to the right and compressed. The posterior section (Fig. 20) showed the hind part of the growth well below the tentorium so compressing the pons that the aqueduct of Sylvius could not be identified.

Histologically, the tumor was composed of areas of small lymphoid cells, mingled with areas of larger, spherical cells (Fig. 21). Connective tissue is especially abundant in the areas around the chief masses of lymphoid cells (Fig. 22). Neuroglia cells and neuroglia fibrillae are occasionally seen (Fig. 24). Blood vessels are rare. The large spherical cells have large nuclei and scanty cytoplasm. In the cytoplasm of the cells may be seen diplosomes, sometimes more than one in a single cell. Numerous mitochondria are also found. Many of the cells give off short stubby processes (Fig. 23).

The tumor, in short, is composed essentially of pineal parenchyma cells with an abundant connective tissue stroma, infiltrated with lymphoid cells.

The following case presented an obscure and complicated clinical picture owing possibly to a double lesion, for in addition to the pineal

Fig. 19 (Case 6).—Papillae covered with squamous epithelium. Hematoxylin and eosin stain; X 80.

tumor the patient had a growth of different histologic character in the optic chiasm,[8] which caused a diabetes insipidus with great emaciation.

CASE 8.—*Double tumor; pinealoma of adult type with glioma of optic chiasm; symptoms of the areas contiguous to both the pineal and hypophysial glands; emaciation; diabetes insipidus.*

History.—On Jan. 31, 1922, Moses P., aged 13, referred by Dr. S. Block of Brooklyn, was admitted to the hospital, complaining of loss of appetite, weakness and headache. His family and past histories were unimportant. The present illness began three years before admission with occasional severe headaches and vomiting. Two years later, there was noticed a decided increase in thirst and a marked polyuria. Eight months before admission the headaches became con-

8. Case reported from this aspect by P. Martin and H. Cushing, Arch. Ophth. **52**:209-241, 1923.

tinuous and vision commenced to fail. Loss of appetite was marked and vomiting was frequent. He had lost 40 pounds (18 kg.) in weight. With the onset of these symptoms, the eyes were examined, disclosing a bilateral choked disk and what was said to be a bitemporal hemianopsia. A right subtemporal decompression had been performed three months before admission, and two weeks later the same procedure was carried out on the left side. Each operation was said to have given some slight relief of headache.

Physical Examination.—The child showed an extreme degree of emaciation and enfeeblement. The skin was pale and dry. Neither axillary nor pubic hair was present. The genitalia were infantile, and one testicle was undescended. The eyegrounds showed what was taken by us to be a bilateral primary atrophy.

Fig. 20 (Case 7).—Coronal section looking forward; shows hind portion of tumor compressing pons.

Conjugate movements of the eyeballs above the horizontal plane were impossible. Contrary to what might have been expected, the roentgenogram of the skull showed a normal sella.

Treatment and Course.—During his stay in the hospital, the boy ran a subnormal temperature with an accelerated pulse. He had a definite polyuria, averaging 2,000 c.c. a day, while his fluid intake ranged about 1,000 c.c. greater in amount. These figures were reduced to normal temporarily by the intranasal administration of pituitary extract. No operation was performed. Death occurred on March 21, 1922, about seven weeks after admission. A complete necropsy was obtained, and performed twenty-one hours postmortem.

Necropsy Findings.—Sagittal section of the brain reached a tumor mass in the region of the pineal body and another in the hypothalamic region (Fig. 25). The hypothalamic tumor, which might easily from its gross appearance have been

regarded as an implantation from the larger tumor, proved to be a glioma of the chiasm (on which basis it has already been reported). The pineal tumor, on the other hand, had a different histologic structure.

Microscopic examination showed it to be composed of a mass of adult pineal parenchyma cells similar to those shown in Figure 32, with a connective tissue reticulum (Figs. 26 and 27). Blood vessels were rare. The cells were large and spherical, occasionally with branched processes. They contained large, spherical nuclei. The cytoplasm was scanty and usually contained a centrosome in the form of a diplosome. Occasionally, short rod-shaped blepharoplasts were seen and numerous mitochondria. Areas of smaller cells were seen here and there.

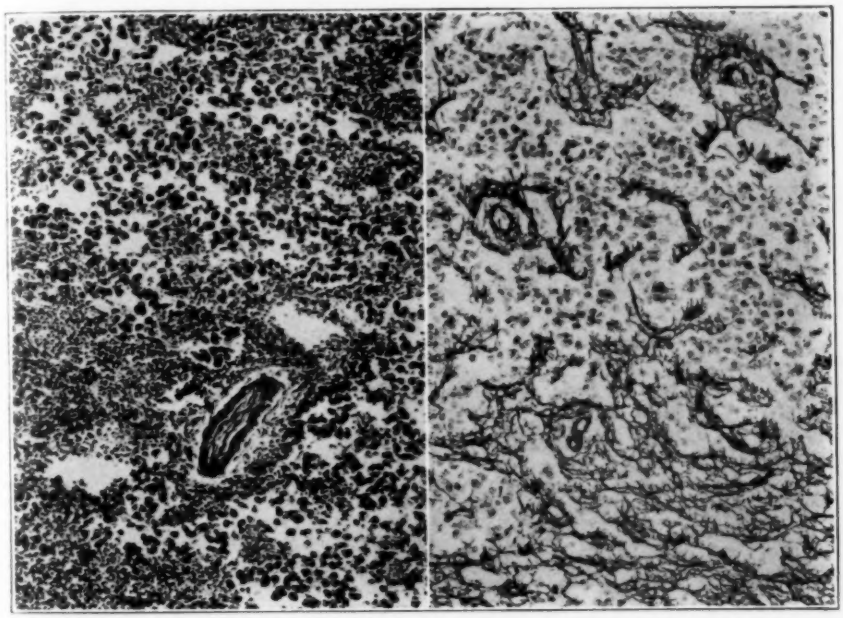

Figure 21 Figure 22

Fig. 21 (Case 7).—General structure of the tumor. Note the large pineal parenchyma cells and the lymphoid cells of the connective tissue reticulum. Hematoxylin and eosin stain; X 80.

Fig. 22 (Case 7).—Connective tissue reticulum. Perdrau's method; X 80.

These cells resemble lymphocytes, having small round nuclei and practically no cytoplasm, and lie in the connective tissue reticulum. No glia cells nor neuroglia fibrillae were seen. The tumor was composed essentially of pineal parenchyma cells, which had not developed abundant processes.

The hypophysis was normal macroscopically and microscopically. The testes were under-developed, the seminiferous tubules containing only a single layer of cuboidal epithelium. There was tuberculosis and fibrous pleurisy of the right lung. A small accessory suprarenal was found. The other organs, including the prostate, carotid body, parathyroid, thyroid, thymus, suprarenal, pancreas and spleen appeared normal.

The general condition of this patient at the time of admission would have precluded any attempt at operative therapy even if such a course had been clear. In view of the outspoken evidences of a suprasellar lesion, this region would have been explored if any operation had been undertaken. So far as his pineal symptoms were concerned, not only was there a total absence of any form of precocity, but, if anything, the reverse of such a picture existed. It may be assumed that his infantile appearance was due to the growth in the neighborhood of the hypophysis which was doubtless responsible for the polyuria (diabetes insipidus).

Figure 23

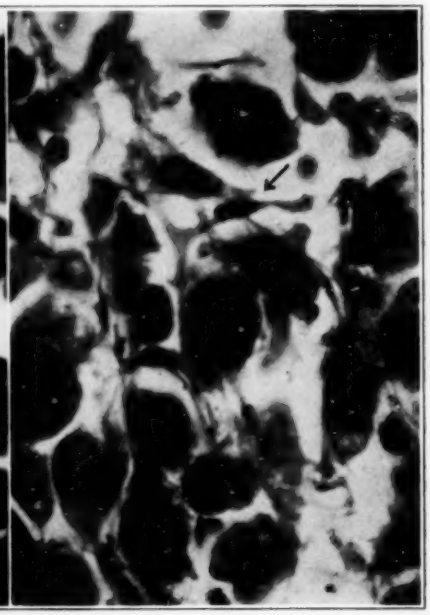

Figure 24

Fig. 23 (Case 7).—Stubby processes of the pineal parenchyma cells. Walter preparation; X 850.

Fig. 24 (Case 7).—Photomicrograph of tumor. Phosphotungstic acid hematoxylin stain; X 850. Shows well the different types of cells occurring in the normal pineal body. At tip of arrow is a connective tissue cell with elongated nucleus. Just to the left and above is a somewhat larger, vesicular, but also elongated nucleus of a neuroglia cell. The huge spherical nuclei of the pineal parenchyma cells are all around.

In the similar case reported by Klaproth, the hypophysis was normal, and in our patient the microscope revealed no changes in the pituitary cells. Had not a careful comparative histologic study been made of the two tumor masses, they would in all probability have been regarded as one lesion with a third ventricle implantation so common in cases of pineal tumor.

In the following case, as in the foregoing, there was a double lesion, a small pineal tumor which could hardly have given symptoms, combined with a large ventricular tumor causing diabetes insipidus.

CASE 9.—*Small pinealoma in association with a large vascular glioma (?) of the third ventricle causing signs in the contiguous areas and diabetes insipidus.*

History.—On July 21, 1922, George H. C., aged 17, was transferred from the medical service with the diagnosis of brain tumor. The family and past history were negative. In August, 1921, the patient started suddenly to show a marked grade of polydipsia and polyuria, amounting to 6 or 7 liters a day. He was admitted to the medical wards. Because of a double plus blood Wassermann

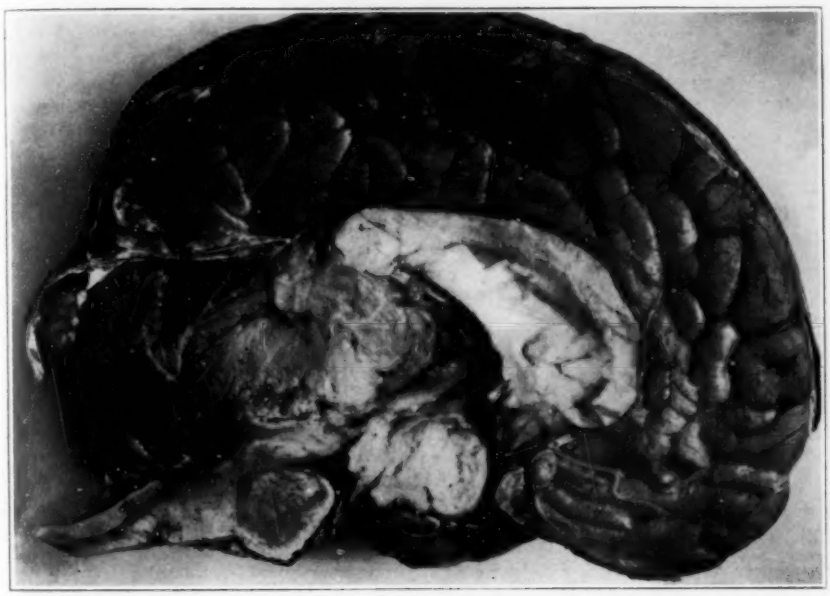

Fig. 25 (Case 8).—Note that the tumor in the infundibulum is one of distinctly different nature instead of an implantation growth from the pineal tumor as in Case 4.

reaction, a rigorous antisyphilitic regimen was instituted and carried out for several months. There was no subsidence of the polyuria, and he became progressively emaciated. Finally, he began to have frontal headaches, double vision and vomiting. These symptoms led to his transfer.

Physical Examination.—The patient was a markedly emaciated young man with a curious brownish pigmentation of the skin. The temperature was subnormal. The blood pressure was: systolic, 90; diastolic, 60. The basal metabolism was —38. He was dull, listless and feeble. The neurologic signs of chief moment related to his diabetes insipidus and to his oculomotor mechanism. The pupils which at first were equal and sluggish to light and in accommodation, within a few days became variable as to size and quite fixed. Convergence was impossible, and he developed first a ptosis of the left eyelid and then of the right.

The fundi and visual fields were normal. The deep tendon reflexes were hyperactive on both sides, and there was a positive Babinski sign on the right.

Treatment and Course.—A diagnosis of tumor of the third ventricle was made and no operative therapy advised. On Aug. 24, 1922, the patient's temperature dropped to 95 F. and then rose steadily to 103 F., when death occurred. A complete postmortem examination was made.

Necropsy Findings.—Sagittal section of the brain revealed a small tumor of the pineal body and a much larger tumor of the third ventricle (Fig. 28). The latter was entirely independent of the pineal tumor and will be made the subject of a special communication. The pineal tumor proved to be more sharply delim-

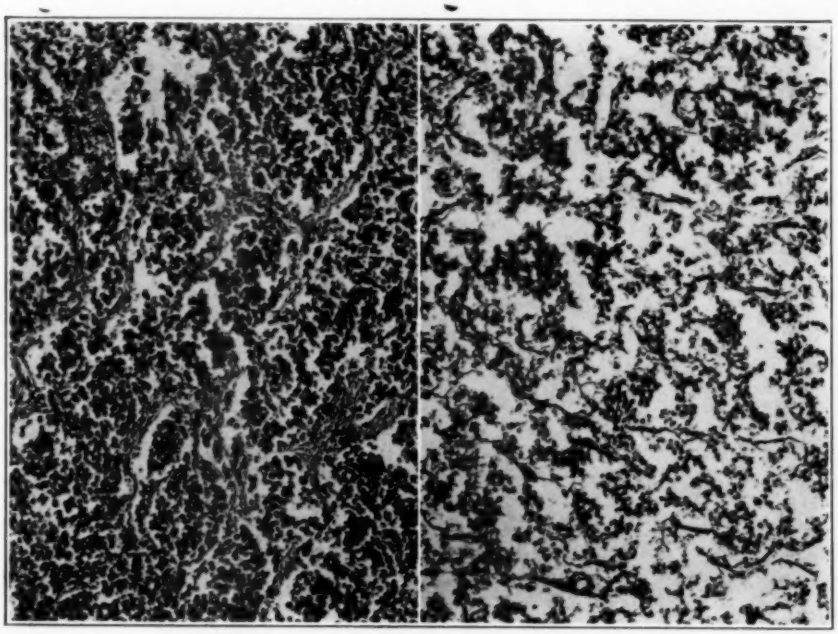

Figure 26

Figure 27

Fig. 26 (Case 8).—Note the large pineal parenchyma cells and the lymphoid cells of the connective tissue reticulum. Ethyl violet-orange G stain; X 80.

Fig. 27 (Case 8).—To show the connective tissue reticulum. Perdrau's method; X 80.

ited than appears from the gross specimen. It was composed of large spherical cells, more or less alveolated by a connective tissue stroma (Figs. 29 and 30). Neuroglia fibrillae in large bundles were present in the peripheral parts of the tumor, but were rare in the more central parts (Fig. 31). Areas of small lymphoid cells were rather frequent. The large spherical cells, which mainly composed the growth, had a large, vesicular, spherical nucleus and scanty cytoplasm, which gave off numerous processes running among the other cells and into the connective tissue septums (Fig. 32). In the cytoplasm of most of the cells could be seen a centrosome in the form of a diplosome.

The tumor was composed essentially of pineal parenchyma cells with a connective tissue and neuroglia framework.

The tumor of the third ventricle was a vascular growth, in structure entirely different from the pineal tumor. It will be described in a separate communication.

Besides a bilateral bronchopneumonia, the internal organs, including hypophysis, parathyroids, thyroids, thymus, suprarenals, pancreas, spleen, prostate and testes, appeared normal.

The outstanding features of the preceding case were the diabetes insipidus, emaciation and the progressive involvement of the oculomotor mechanism, due in all probability to the ventricular tumor rather than to the small pinealoma which of itself was hardly large enough to have

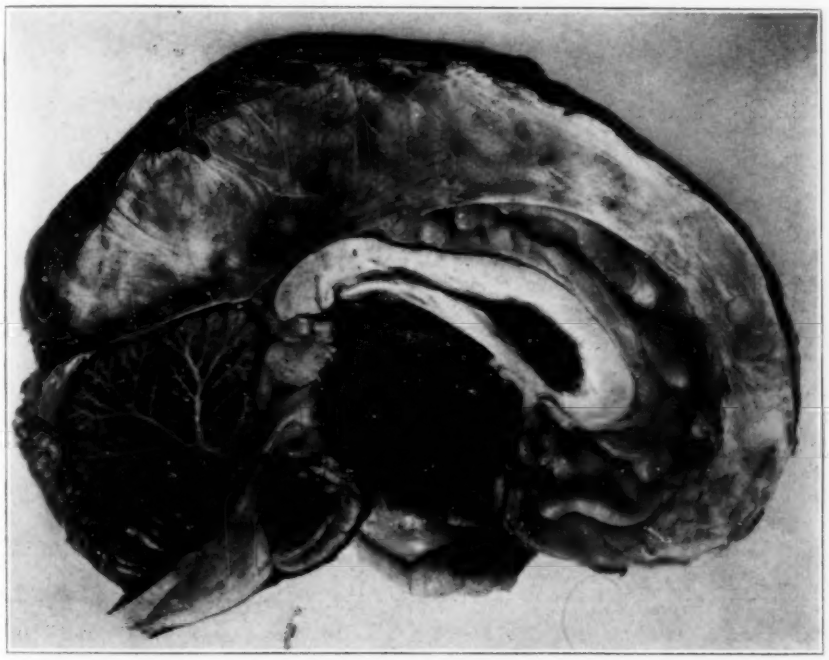

Fig. 28 (Case 9).—Mesial section showing small pineal tumor and large vascular tumor of third ventricle anterior to it.

caused any marked neurologic symptoms. It is perhaps remarkable that the eyegrounds showed no papilledema in view of the size and location of the main growth with the resulting hydrocephalus.

The following case showed many clinical features similar to the foregoing though produced by a single tumor:

CASE 10.—*Pinealoma of adult type with definite localizing neurologic symptoms, hydrocephalus and diabetes insipidus.*

History.—On July 11, 1923, Leslie P., a man, aged 20, was admitted to the hospital, referred by Dr. L. S. Aronson of New York. Neither his family nor past history was of importance with the exception possibly of the fact that he

began to shave later in life than usual. The present illness began in September, 1922, with attacks of supra-orbital persistent headache, radiating to both temporal regions. Within a few months of the onset, there developed polydipsia and polyuria, as well as occasional attacks of projectile vomiting. Soon vision also commenced to fail progressively, until all sight was lost about three weeks before the patient was admitted to the hospital. Bilateral choked disks had been noted in November, 1922, and operation advised, but refused.

Physical Examination.—This disclosed absolute blindness with dilated and fixed pupils. There was bilateral papilledema of 5 diopters, with marked secondary atrophy. Conjugate movements of the eyeballs above the horizontal were

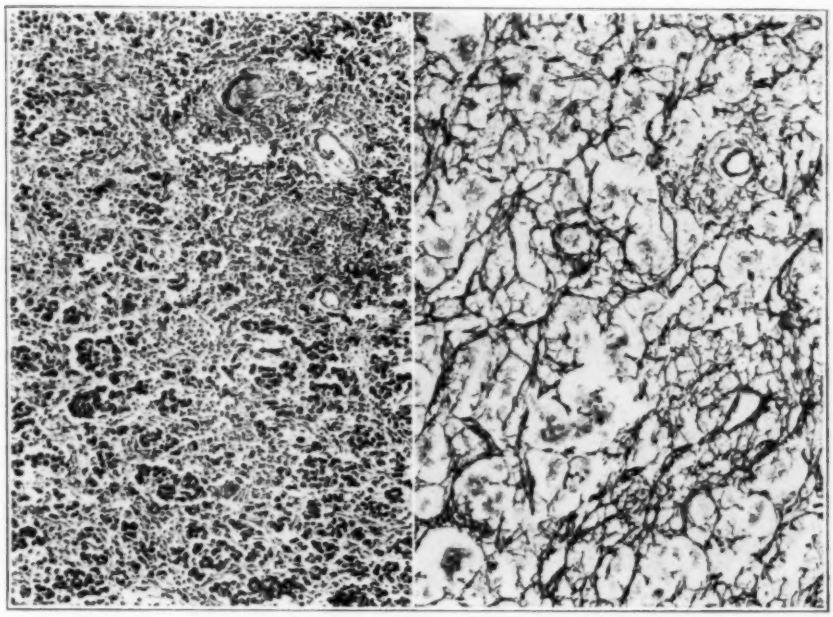

Figure 29 Figure 30

Fig. 29 (Case 9).—Note the large pineal parenchyma cells and the lymphoid cells of the connective tissue reticulum. Hematoxylin and eosin stain; X 80.

Fig. 30 (Case 9).—Connective tissue reticulum. Perdrau's method; X 80.

impossible, and there was a bilateral abducens paralysis. There was coarse nystagmus to the right and left. Hearing was slightly impaired on both sides, with bone conduction greater than air conduction on the left. The external genitalia were small and the pubic hair showed a feminine distribution. The patient was greatly emaciated. A roentgenogram of the skull showed marked convolutional atrophy and erosion of the posterior clinoids. From the time of his admission to the time of his death this patient showed a profound polydipsia and polyuria. The intake averaged between 5,000 and 6,000 c.c., at times going as high as 8,500 c.c. The output averaged 1 or 2 liters less in amount.

Course of Illness.—It was thought that the patient had a tumor involving the third ventricle, but an operation was contraindicated largely because it was

found that he had a coincidental pleurisy with effusion. Cultures from the thoracic fluid were negative and no tubercle bacilli were found. He died following a generalized convulsion, Sept. 3, 1923. A complete necropsy was performed six hours after death.

Description of Tumor.—A sagittal section of the brain showed a growth occupying the position of the pineal body. Its relationships are evident from the illustration (Fig. 33). The tumor was found to be composed (Fig. 34) of irregular islands of large spherical cells separated by bands of neuroglia fibrillae (Fig. 36). Connective tissue was scanty and confined almost exclusively to the walls of the rare blood vessels. The large spherical cells had a scanty cytoplasm and a large vesicular, spherical nucleus. No granules of any kind could be

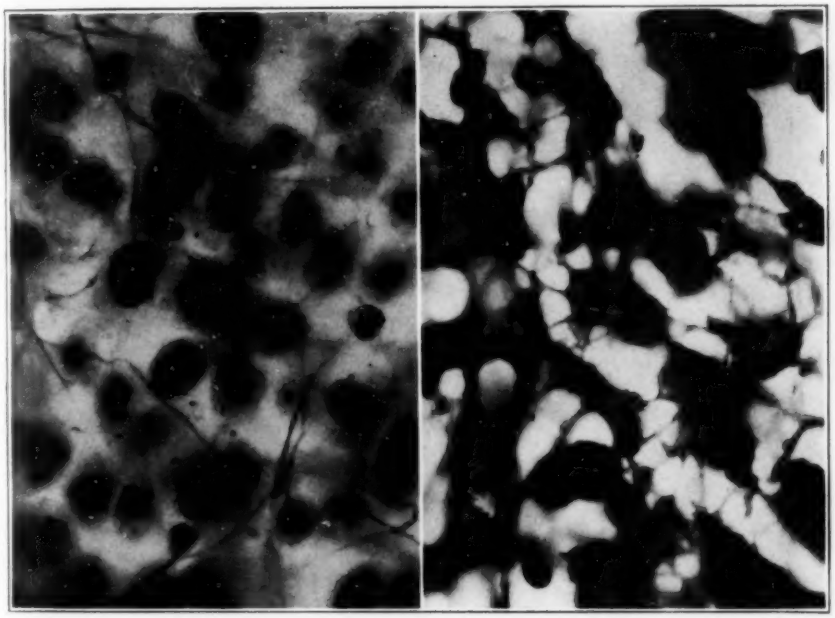

Figure 31

Figure 32

Fig. 31 (Case 9.—Note the neuroglia fibrillae. Hortega's first variant; X 850.

Fig. 32 (Case 9).—Processes of the pineal parenchyma cells. Walter's method; X 850.

demonstrated in the cytoplasm. The cell boundaries were sharp and heavily stained. Many cells gave off processes which were for the most part unbranched (Fig. 35). Lymphoid cells were found in small areas of the connective tissue reticulum. The neoplasm, in short, was composed essentially of pineal parenchyma cells, with a stroma of neuroglia.

General Examination.—This showed miliary tuberculosis of the lungs, spleen, liver and bronchial lymph nodes; tuberculous pleuritis, chronic fibrous peritonitis, and left hydrothorax. The other internal organs appeared normal.

Here again the interesting features for diagnosis were, as in the previous case, polyuria, emaciation, involvement of the oculomotor

centers, as well as beginning pressure on the inferior colliculi, denoted by impaired hearing and in the later stages by bilateral spasticity. No ready explanation of the diabetes insipidus can be offered. The third ventricle (Fig. 33) was greatly dilated, but this is a common complication of tumor without any polyuria. Had the diagnosis of pineal tumor been definitely made, we should have expected to find an implantation in the infundibulum, and it is possible that there may have been one

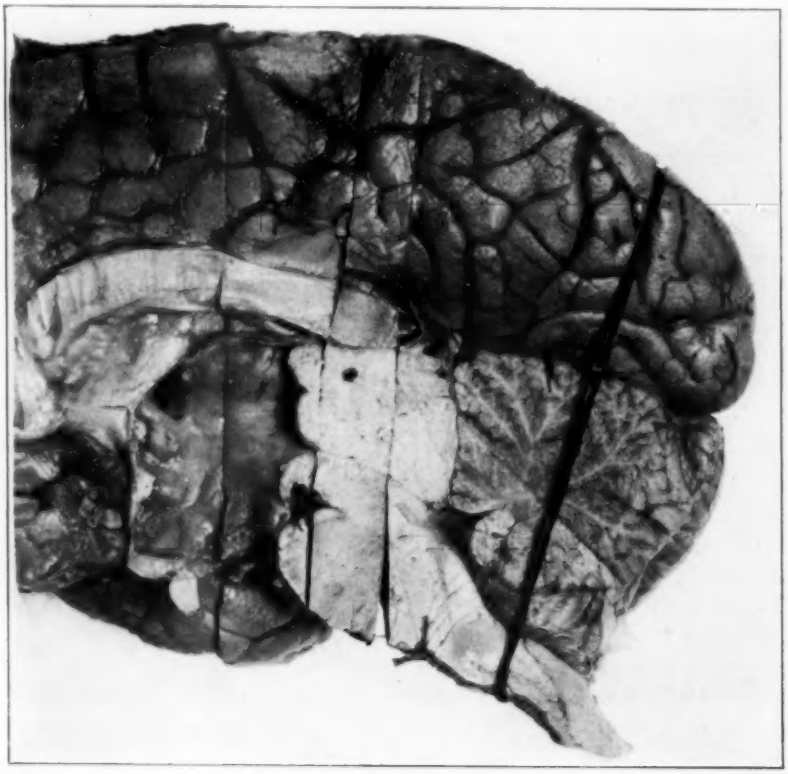

Fig. 33 (Case 10).—Note the trapezoidal shape of the tumor and its line of demarcation from the compressed corpora quadrigemina.

dislodged in handling the brain, which was abundantly sectioned, as the illustration shows.

The following case was at first puzzling because of the definite trigeminal sensory loss, but ventriculograms later showed symmetrically dilated ventricles:

CASE 11.—*Greatly degenerated pinealoma of spongioblastic type in a young girl, with characteristic signs in the contiguous area, and hydrocephalus.*

History.—On Jan. 9, 1924, Mary St. J., aged 15, was referred by Dr. H. H. Bard, of Pittsfield, Mass., with the diagnosis of intracranial tumor. The past

history brought out the fact that her menses had become established at 13 years of age and had been regular until the onset of the present malady. This began six months before her admission, with severe frontal headaches which, however, had become less severe and persistent of late. Failing vision accompanied the headaches and progressed to blindness in the right eye. Diplopia was present before she became blind. During the three months before admission she had gained 30 pounds (13.6 kg.) in weight. Walking had become slightly unsteady.

Physical Examination.—The patient was somewhat obese, with a large head. There was marked bulging over both temporal regions, where the bone was so thin that it could easily be indented with the finger (craniotabes). The eye-grounds showed bilateral choked disks of 6 to 7 diopters, with large amounts of

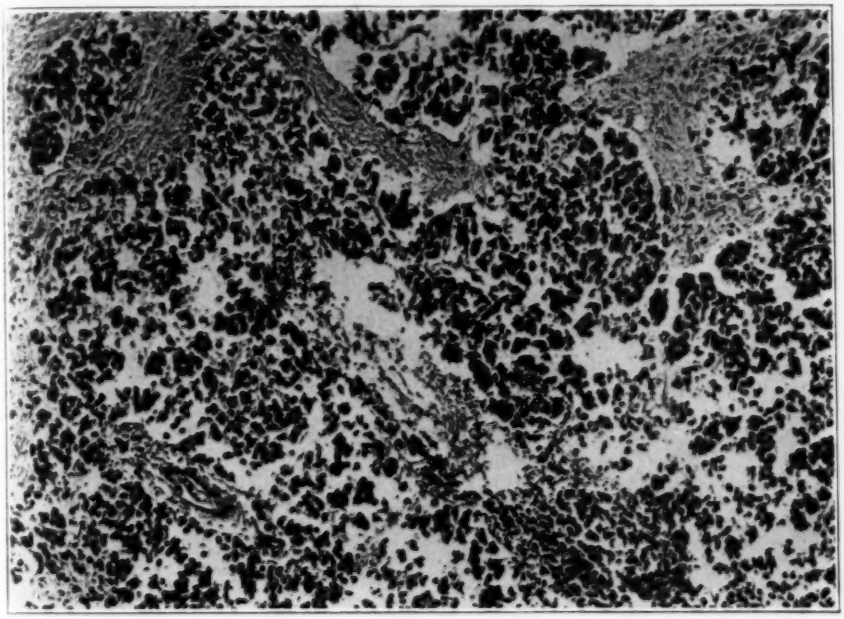

Fig. 34 (Case 10).—Pineal parenchyma cells in large groups. In the lower center are lymphoid cells of a scanty connective tissue reticulum. The broad bands above consist of neuroglia cells and fibrillae. Hematoxylin and eosin stain; X 80.

new tissue and many hemorrhages. She was blind except for the retention of light perception with the left eye. Convergence was poor. There was marked weakness of the right internal rectus. Conjugate movements of the eyeballs were impossible above the horizontal plane. There was hypesthesia of the right side of the face with loss of the corneal reflex on that side. The deep reflexes were greatly exaggerated on both sides in all extremities. Romberg's sign was positive. Her basal metabolic rate was 30 below normal. The roentgen ray showed no localizing signs of tumor.

Treatment and Course.—In view of her definite trigeminal sensory loss, Dr. Cushing explored the right subtemporal region on Jan. 22, 1924, but disclosed

only an internal hydrocephalus, which was subsequently shown by ventriculograms to represent a symmetrical dilatation. Realizing that the condition must be due to a central (pontile ?) lesion with hydrocephalus, an effort to give some pressure relief was undertaken by Dr. Cushing on January 29. The third ventricle was exposed by means of a frontal osteoplastic flap and elevation of the right frontal lobe. The bulging dilated ventricle was opened widely in the hope that this would permit the escape into the arachnoid spaces of the ventricular fluid, and afford some relief of the hydrocephalus. There was, however, no improvement after this procedure, and the patient died about three weeks later after a gradual decline. Permission for an examination other than what could be gained

Figure 35

Figure 36

Fig. 35 (Case 10).—Processes of the pineal parenchyma cells. Walter's method; X 850.

Fig. 36 (Case 10).—Neuroglia fibrillae. Heidenhain's iron hematoxylin; X 850.

through the operative wound was refused. The hind-brain was removed in this way six hours postmortem.

Pathologic Findings.—The restricted necropsy made it necessary to remove the brain in fragments, and the cerebral hemispheres were practically destroyed in so doing, since there was a marked internal hydrocephalus. The hind-brain, however, was removed more or less intact. It showed (Fig. 37) a circumscribed tumor mass a little more than 2 cm. in diameter in the position of the pineal body. The aqueduct of Sylvius was patent, and separating it from the tumor was a thin layer of nervous tissue.

Microscopically, the tumor was found to be extensively degenerated throughout. Only pyknotic nuclei, a few small blood vessels and coarse neuroglia fibrillae

could be made out. Colloid and hyaline material made up the rest of the tumor (Figs. 38 and 39). The growth must originally have been composed of embryonic neuroglia cells.

The following, the last case in the series,[9] resembled clinically a cerebellar tumor. The course of the patient's illness was extremely rapid, and he died before any operative intervention.

CASE 12.—*Pinealoma of adult type in a young man with supposedly cerebellar symptoms.*

History.—On April 7, 1924, John A. C., aged 24, referred by Dr. A. S. Hartwell of Norwood, Mass., was transferred from the medical to the surgical service. His present illness began about March 1, 1924, with gradually increasing headaches,

Fig. 37 (Case 11).—Relations of the tumor to cerebellum and brain stem (limited necropsy).

associated at times with vomiting and dizziness. He was admitted to the medical service on March 13, but was discharged after several days' observation with no definite diagnosis, although brain tumor was suspected. He returned on April 4, with a story of having become unsteady in gait during the interim, and of dizziness with vomiting on any change of position in bed. The fundi on this readmission now showed an early choking, the disks being elevated at least 1 diopter. A nearly complete paralysis of the left abducens was present, and convergence with the right eye was impossible. He had also some slight left lower facial weakness. There were well marked hypotonia and dysmetria of both upper and lower extremities; also marked suboccipital tenderness with cervical rigidity. He had in addition some deglutitory difficulty and dysarthria.

9. One other example has been disclosed since these notes were made, making thirteen in 1,053 verified tumors.

He had evidently gone down hill rapidly since his previous admission owing, as was presumed, to a cerebellar tumor. Before an operation could be performed, he died, on April 10, of sudden respiratory failure.

Necropsy Findings.—An examination of the head only was permitted; this was made eight hours postmortem. It disclosed a small roundish tumor mass sharply delimited from the brain tissue, about 1.5 cm. in its greatest diameter, occupying the position of the pineal body (Fig. 40).

The neoplasm histologically was found to be composed of large spheroidal cells separated into masses by heavy, branching connective tissue septums (Figs. 41 and 42). Occasional fibrillae of neuroglia cells were identified by specific stains (Fig. 44). The large cells had round vesicular nuclei. Many were true

Figure 38

Figure 39

Fig. 38 (Case 11).—Appearance of tumor at low magnification. Hematoxylin and eosin stain; X 850.

Fig. 39 (Case 11).—Coarse neuroglia fibrillae. Hortega's first variant; X 850.

giant cells with several nuclei. In addition, there were many areas remote from the blood vessels in which the nuclei had entirely disappeared from the cells, leaving a large vacuole in the center. Such areas may be identified in Figure 41. The cytoplasm of these cells contained centrosomes in the form of diplosomes or occasionally triplosomes, fine granular mitochondria, and often a profusion of coarse granules of doubtful nature. Many of the cells gave off short processes which ended in typical end bulbs (Fig. 43).

The tumor therefore was composed essentially of pineal parenchyma cells, in a fairly advanced stage of development, but definitely pathologic. In addition, there were occasional neuroglia cells and a heavy connective tissue framework. The growth was a pinealoma of adult type.

THE NEUROLOGIC SYMPTOMATOLOGY

In the introduction to this paper, it was stated that the three points to be specially considered in relation to pineal tumors were: (1) their neurologic, (2) their endocrinologic, and (3) their histopathologic aspects. The neurologic findings may be dismissed with a few remarks. They concern chiefly the oculomotor apparatus, the auditory connections in the inferior colliculi, spasticity of the limbs, and evidences of pressure on the cerebellum itself or on its tracts.

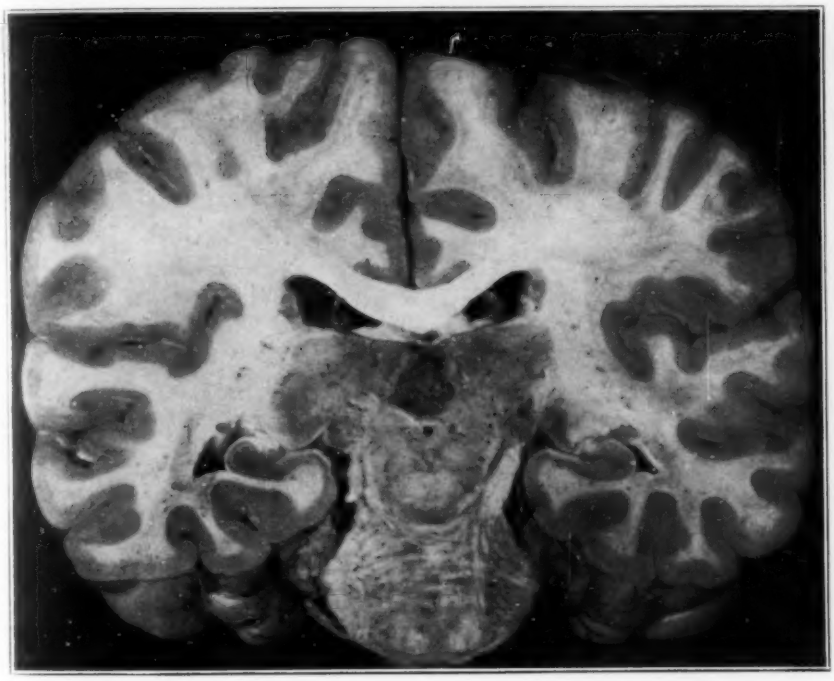

Fig. 40 (Case 12).—A chance coronal section passed nearly through the center of small pineal tumor.

To state this in detail, nine of the twelve patients showed manifestations of oculomotor involvement, the most characteristic being loss of conjugate movement of the eyeballs above the horizontal plane, a finding which was present in four cases. In addition, there were noted unequal pupils (three cases); diplopia (three cases); paralysis of convergence (two cases); paralysis of right internal rectus (two cases); bilateral ptosis (one case); fixed pupils without blindness (one case); and lack of parallelism of the eyeballs (one case).

Spasticity of the limbs was bilateral and marked in five of the twelve cases, and moderate in one other. In two instances, Babinski's sign was

positive on both sides, and in another on the right only. Symptoms implicating the cerebellum were manifested in nine cases, being of sufficient grade to warrant a suboccipital operation in four of the patients and a threat of the same procedure in others.

In respect to other features, three patients showed bilateral abducens paralysis, and three showed palsy of this nerve on a single side. Five cases showed some degree of central deafness, this being complete in three cases, complete on one side in another and mild on both sides in

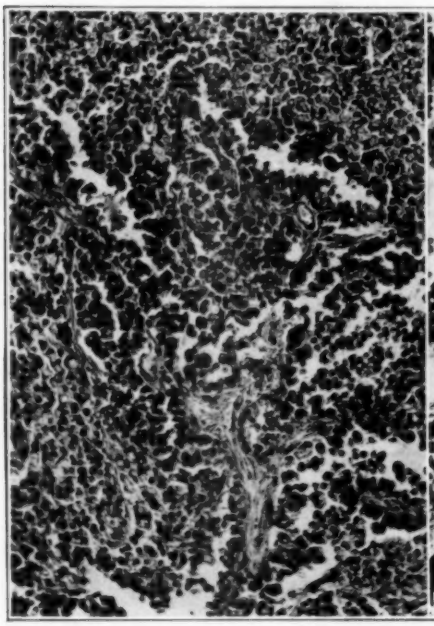

Figure 41

Figure 42

Fig. 41 (Case 12).—General appearance of the tumor. Hematoxylin and eosin stain; X 80.

Fig. 42 (Case 12).—Note heavy connective tissue septums. Perdrau's method; X 80.

a third. One patient showed hypesthesia of one side of the body, while in another there was loss of sensation of one fifth nerve. Polyuria in four cases was well marked.

In retrospect, it would seem that the correct diagnosis should have been made in the two cases of outspoken precocity and at least strongly suspected in the four other patients who showed a lack of conjugate movements of their eyeballs upward in addition to other signs of a growth near the pineal gland.

These cases can have few surgical bearings, an attempt to drain the hydrocephalus being perhaps the only logical procedure, although attempts have been made to extirpate teratomas. The latter may in some instances be differentiated from the pinealomas by calcification shown in the roentgenogram, but in our only case of teratoma no calcification was present.

THE ENDOCRINOLOGIC QUESTION

In five of the cases in the series (Nos. 3, 4, 5, 6 and 8) the patients were children, in each of whom at necropsy a tumor of the pineal body

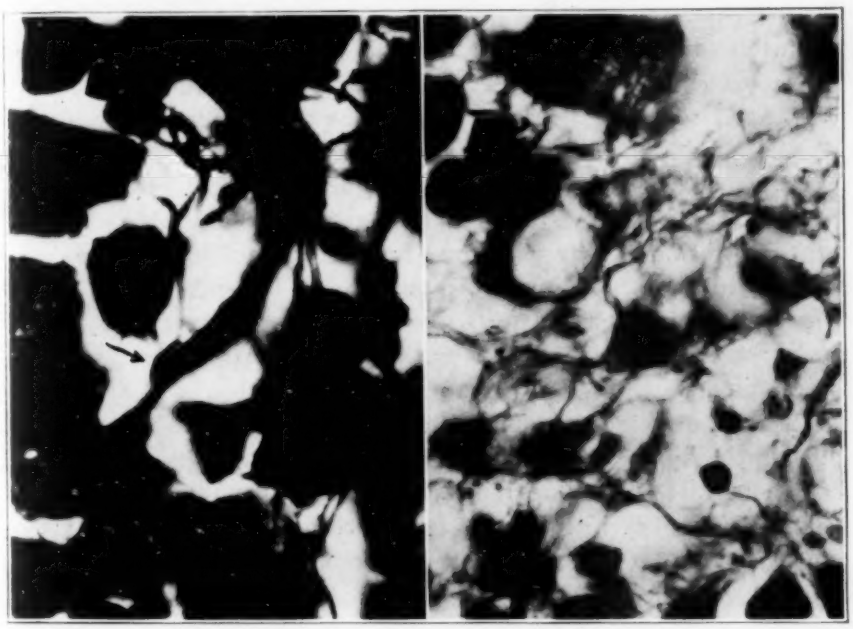

Figure 43 Figure 44

Fig. 43 (Case 12).—A typical end bulb of a pineal parenchyma cell. Walter's method; X 850.

Fig. 44 (Case 12).—Neuroglia cells and fibrillae. Heidenhain's iron hematoxylin; X 850.

was disclosed before the age of puberty. Neurologically, they all showed signs of increased intracranial pressure of marked degree, together with more or less unmistakable evidences of pressure on structures in the area near the pineal body. In two instances, limitation of conjugate movements of the eyeballs in the vertical plane was recorded and bespoke direct implication of the region of the cerebral peduncles, while in a third, bilateral deafness denoted involvement of the inferior colliculi.

As to the symptoms which have been supposed to be due to a disturbance of function of the epiphysis, we have the following data. Three of the patients showed evidence of mental precocity, this being well marked in two of them. Three patients showed decided bodily overgrowth for their respective ages. In Case 2, there was apparently full sexual maturity at the age of 11 and in Case 3 there was probably some overdevelopment of the external genitalia, although this was far from the outspoken type which has been described as occurring in very young children. It must be said therefore that in only two of these five cases was the picture of pubertas praecox outspoken (Cases 4 and 6), being most complete in the patient (Case 4) whose case was formerly reported from this clinic by one of us (Horrax).

In considering the prepubertal cases as a whole, what, if anything, can be said regarding a relation between the pineal body and precocious development? So far as the clinical diagnosis of a pineal tumor is concerned, the association of mental, physical and sexual precocity with intracranial pressure symptoms remains as important a syndrome today as it did when originally urged by Frankl-Hochwart [7] in 1909. It may be said, further, that precocity in any two of the spheres which have been mentioned, combined with signs of increased intracranial tension and implication of the corpora quadrigemina, is strong evidence of a pineal tumor.

When, however, we come to the question as to whether sexual or other precocity is due to a disturbed function of the pineal body, we are on much less certain ground. Krabbe, in his admirable treatise, points out that before coming to any conclusions six possible elements must be taken into account: (1) *secretion of a substance from the tumor itself;* (2) *destruction of the pineal body;* (3) *destruction of neighboring brain centers;* (4) *distant action on the hypophysis;* (5) *the constitutional element which expresses itself in the development of the teratomas and* (6) *deficient investigation of the other organs.* These elements may be separately considered.

(1) In regard to the first of them, the opinion has been maintained, especially by Askanazy,[10] that only teratomas of the pineal body were associated with precocity. To substantiate this opinion, Askanazy, in 1920, reexamined the tumors other than teratomas which had been reported in patients showing pubertas praecox, and concluded that all cases, however originally designated, were in reality teratomas. In this investigation, however, he overlooked the case reported from this clinic

10. Askanazy: Die Zirbel und ihre Tumoren in ihrem funktionellen Einfluss, Frankf. Ztschr. f. Path. **24**:58, 1920.

by Horrax (1916) and the case of Skoog (1918),[11] both of which were unquestionably tumors composed largely of undifferentiated pineal cells, and reported as such. Thus, although the theory of Askanazy is overthrown, the possibility of a substance being elaborated by what have been called "adenomas" of the pineal body (the "pinealoma" of Krabbe) must still be considered. This possibility we feel does not exist, because from our histologic studies, the epiphysis does not contain glandular cells, and therefore adenomas of this organ, unlike the thyroid, hypophysis and suprarenal, probably do not occur.

(2) There seems to be little doubt that retrogressive and degenerative changes usually occur in the pineal body about the time of puberty, although they may begin at any age from 2 years to adult life, and it is a natural assumption that the structure exercises until that time some check on the appearance of the secondary sex characters. However, the experimental destruction of the pineal body in young animals has not been followed by sufficiently clear-cut evidence of resultant sexual precocity to justify any definite conclusions in the matter. Krabbe, moreover, has described a case of complete destruction of the pineal body in a child without pubertas praecox.

(3) In regard to Krabbe's third element, he describes a boy who showed typical pubertas praecox at 18 months of age, and at necropsy was found to have a tuberous sclerosis of the brain and a practically normal pineal body. This observation lends some support to the hypothesis that centers within the brain may be responsible for pubertas praecox. However, the numerous cases of tuberous sclerosis without this syndrome make the hypothesis improbable.

(4) Distant action on the hypophysial region may account for polyuria and adiposity in these patients, but hardly for macrogenitosomia praecox.

(5) That there should be some special constitutional element associated with the teratoma capable of provoking early precocity seems most improbable, for the syndrome in question, although it seems to be more common with the teratomas, is not limited exlusively to lesions of this histologic type.

(6) As complete necropsies were not carried out on many of the cases reported as showing the so-called pineal syndrome, it is impossible to state whether such organs as the suprarenals or the endocrine elements of the testicles were in any way abnormal. It is well known that pubertas praecox may occur with tumors of the suprarenals or testes.

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11. Skoog, A. L.: Pineal Gland Neoplasms: Report of a Case, New York M. J. **107**:1199, 1918.

Some factor, concerning which our information at present is lacking, may furnish the key to unlock the mystery. However, it must be remembered that even were changes found in these other glands, it would not greatly alter our present conception of the production of pubertas praecox; for it would be difficult to tell whether these changes were primary or secondary to the pineal lesion—a decision indeed which is sometimes difficult to make in certain pituitary disorders which show associated polyglandular alterations. About the best we can do at the

All Cases of Pineal Tumor in Patients Showing "Pubertas Praecox."

Year	Author	Age	Sex	Clinical Data	Type of Tumor
1896	Gutzheit	7¾	M	Marked development of pubic hair and public folds	Teratoma
1899	Ostreich and Slawyk	4	M	Large mammary glands with colostrum; penis, 9 cm. long; pubic hair, 1 cm. long; testicles large	Psammosarcoma
1899	Ogle	6	M	Large penis, abundant pubic hair; masturbation	Alveolar sarcoma*
1903	Holzhauer	4	M	Large external genitalia; bodily overgrorwn	Alveolar sarcoma*
1909	Frankl-Hochwart	5½	M	Adult sized genitalia with pubic hair; changed voice; bodily overgrowth; mentally precocious	Teratoma
1910	Raymond and Claude	10	M	Development of pubic hair; precocious mentality	Glioma*
1911	Bailey and Jelliffe	12	M	Bodily overgrowth very rapid; adiposity	Teratoma
1913	Hijmans van den Bergh.	8	M	Overdevelopment of genitalia; pubic hair	Teratoma
1915	Odermatt	5	M	Large genitalia; pubic hair	Teratoma
1916	Horrax	12	M	Bodily overgrowth; adult genitalia; pubic and axillary hair; changed voice	Adenoma*
1918	Skoog	9	M	Rapid development of sexual organs and functions; genitalia as large as boy of 16	Compound pineal gland type*
1919	Boehm	9	M	Large genitalia; pubic hair; changed voice; mental precocity	Teratoma
1921	Lereboullet	12	M	Changed voice; adult type of pubic and axillary hair; genitalia of adult size	Teratoma
1924	Horrax and Bailey	3	M	Bodily overgrowth, mental precocity and probably overlarge genitalia	Teratoma

* These tumors, from the descriptions, we feel sure are identical with our pineaiomas of adult type. In the case of Ostreich and Slawyk, the description is too brief to permit an opinion to be formed of the nature of the tumor.

moment is to say that pineal tumors, particularly teratomas in male children, before puberty may be associated with pubertas praecox, whereas hypophysial tumors, particularly the pharyngeal pouch tumors, in children of both sexes provoke a reverse condition, with infantilism.

A table of the reported cases (so far as known) of pineal tumors in patients below the age of puberty showing premature development—mental, physical and genital—is appended.

In regard to the cases of pineal tumor in children, it may be said that extremely few have been reported in which Pellezzi's syndrome was

present and the lesion later verified by necropsy. In 1911, Pearce Bailey and Jelliffe [12] collected sixty verified cases of pineal tumor in patients of all ages, including one personal case which was reported in full. Of these sixty patients, twenty were below the age of puberty, and seven of these showed marked evidence of premature adolescence. In the other thirteen, premature changes were either not present or not recorded. Another case was reported in 1913 by Hijmans van den Bergh [13] and in 1916 one of us (Horrax)[6] reported three cases of pubertas praecox from Dr. Cushing's clinic, in one of which a pineal tumor was likewise verified. Since that time, the literature contains a few additional verified cases in preadolescent patients with somatic changes ascribed to a disturbed function of the pineal body. A summary of their salient features follows:

Case of Odermatt,[14] *1915.*—A child at 5 years of age acquired pubic hair and had frequent erections. At ten years, he showed symptoms of increased intracranial pressure, and during this year there was a general rapid overdevelopment of the entire body, with change of voice to the deep masculine, adult type. Death occurred in this year, and at necropsy a teratoma of the pineal was disclosed.

Case of A. L. Skoog, 1918.[11]—A boy of 9 years began having symptoms of intracranial pressure and coincidentally showed enlargement of the external genitalia with abnormal development of the sexual functions comparable to that of a boy of 16. Necropsy showed a large tumor of the pineal body classified as the "compound pineal type."

Case of E. Boehm, 1919.[15]—A boy of 9 years had shown premature sexual development for a year. His voice had already changed to the deep adult type, and his external genitalia were of adult size with pubic hair present in abundance. At necropsy a teratoma of the pineal body was found.

Case of Lereboullet, Maillet and Brizard, 1921.[16]—A boy, aged 12 years, had an adult voice, a growth of hair on the face, pubes, and axillae, and a rapid development of the external genitalia to adult proportions. His height was 149 cm. (normal for his age, 136 cm.). Necropsy showed a pineal teratoma.

The following case is of interest because of the fact that although a pineal tumor was demonstrated postmortem, the child, instead of having genital precocity, showed an infantile development.

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12. Bailey, Pearce; and Jelliffe: Tumors of the Pineal Body, Arch. Int. Med. **8**:851-880 (Dec.) 1911.

13. Van den Bergh, Hijmans, and van Hasselt: Tumor glandulae pinealis sive epiphysis cerebri, Nederlansch. Tijdschr. v. Geneesk. **1**:1271, 1913.

14. Odermatt: Zur diagnostik der Zirbeldrüsentumoren, Inaug. Diss., Zurich, 1915.

15. Boehm, E.: Zirbeldrüsenteratom und genitale Frühreife, Frankf. Ztschr. f. Path. **22**:121-146, 1919-20.

16. Lereboullet, Maillet and Brizard: Un cas de tumeur de l'epiphyse, Bull. Soc. de Pédiat. de Paris **19**:116-120, 1921.

Case of W. Klaproth, 1921.[17]—A boy, 15 years of age, whose pubic and axillary hair were lacking, had a small scrotum and testicles, although the penis was normally developed for his age. His general appearance was that of an infantile habitus. Necropsy disclosed a teratoma of the pineal body.

In addition to these five cases of tumor, the following example of hypoplasia of the pineal body should be cited.

Case of Askanazy and Brack, 1921.[18]—An idiot girl was observed by the authors from the age of 8 years until her death at 23. At the age of 10, her breasts began to enlarge rapidly. Pubic hair appeared during the same year. At the age of 11½ years, pubic and axillary hair was adult in amount and appearance. The labia majora were well developed. Necropsy (at the age of 23) revealed a small pineal body which contained none of the normal characteristic elements. It was designated as a hypoplasia.

This represents the only instance of a verified pathologic condition in the pineal body of a female subject showing premature adolescence. Marburg's case,[19] which has often been quoted, was also that of a young girl who had a pineal teratoma, but her only constitutional change consisted in adiposity. This might well enough have been due to a secondary hypophysial influence, for any tumor which produces an internal hydrocephalus in childhood is likely to be accompanied by some degree of adiposity.

It will thus be seen that, in all, fourteen tumors of the pineal body verified by necropsy have been reported in children who showed premature development of the primary and secondary characteristics of sex. All of these patients have been boys. In addition, one boy of 15 with a pineal teratoma was shown to have infantile sexual development and one girl with hypoplasia of the pineal has been reported to have had pubertas praecox. In connection with this example of hypoplasia, attention may be called to Krabbe's case of absolute destruction of the pineal body without pubertas praecox.

DISCUSSION OF PATHOLOGIC ANATOMY

It is evident from the foregoing descriptions that many of the tumors showed all the gross architectual features of the normal epiphysis, consisting of parenchymatous and neuroglial cells in varying proportions (the parenchymatous cells always predominating) divided into lobules by a connective tissue and neuroglial framework. Nevertheless, it

17. Klaproth, W.: Teratom der pineal combiniert mit Adenom, Centralbl. f. allg. Path. u. path. Anat. **32**:617-630, 1921.

18. Askanazy and Brack: Sexuel precosität in einer Idiotin mit Hypoplasie der Pineal, Virchows Arch. f. path. Anat. **234**:1-11, 1921.

19. Marburg: Die Klinik der Zirbeldrüsen Krankungen, Ergebn. d. inn. Med. u. Kinderh. **10**:146, 1913.

would be improper to call them hypertrophies, for the cells were always in an earlier stage of development than the normal adult pineal cells. For these tumors the term used by Krabbe, "pinealoma," seems to us best, and we have added "of adult type," for the attempt to reproduce the structure of the adult pineal body is obvious. There is no question that these "pinealomas" arose from the pineal anlage.

The case is not so clear for the tumors of Cases 1, 2, 3, 5 and 11, which were composed of less differentiated epithelial cells. Of these, Case 1 is the least sure since in histologic structure it was different from the others (Cases 2, 3, 5 and 11), and resembled tumors sometimes seen in the cerebral hemispheres. Yet in each of these cases, we have reasons to believe from the microscopic examination that the tumor was outside the neuraxis, although from the gross appearance, it might appear that it arose from the corpora quadrigemina. In Case 2, a section through the entire hemisphere was made to prove the relationships of the tumor. At any rate, in every instance the pineal body was completely destroyed. It is, in our opinion, much more likely that tumors of embryonic cells in this region should arise from the pineal anlage than from the corpora quadrigemina, but we have excluded from our series one such tumor of this region in a child of 5 years, because we could not convince ourselves that it was extracerebral.

It was difficult to find a suitable terminology to employ for these relatively undifferentiated tumors of the pineal anlage, but it was finally decided to call them also "pinealomas" since they arise from the pineal anlage, and to distinguish them by adding "of spongioblastic type" to emphasize the undifferentiated character of the cells. How closely they may reproduce the structure of the pineal anlage may be appreciated from a comparison of Figures 1 and 16.

Besides pinealomas and teratomas, we have found no other types of tumors in our series. Gliomas and sarcomas are theoretically possible, but from an examination of the literature we feel that, although connective tissue or neuroglial tissue may predominate in a certain tumor (case of Raymond and Claude), closer examination will usually reveal some of the other constituents of the pineal body.

Since the pinealomas reproduce, more or less exactly, the structure of the normal pineal body, we must examine for a moment the question whether the structure of these tumors offers any support to the hypothesis that "macrogenitosomia praecox" is the result of the secretory activity of a pineal tumor. We can only answer that we have found no evidence of secretory activity in the parenchymatous cells of the pinealomas. This is consistent with the fact that there is no evidence of secretory activity in the parenchymatous cells of the normal pineal

body. We are entirely in accord with Hortega [4] in saying that we have never observed in any case the existence of granules homologous to the secretory granules of active glands in the protoplasm of the parenchymatous cells of the pineal body. We are unable to accept the possibility of an intervention of the nucleus in the secretion, in the manner supposed by Dimitrova, a phenomenon which would be without parallel in glands known to possess an active secretory activity.

Hortega maintains that if one holds to the exact representation of the facts observed, there is lacking decisive histologic evidence of secretory activity in the pineal body. A similar observation was made by Bailey concerning the hypophysis cerebri.[20]. In fact, this lack of histologic evidence of secretory activity is apparent in the case of almost every gland of internal secretion, as one may learn from a persual of Swale Vincent's recent work.[21]

The histologic changes in a cell generally considered as incidental to secretory activity are: (1) a shift of the nucleus from the base of the cell toward the secreting surface; (2) the reduction in number of secretory granules which gather near the secreting surface; (3) formation of vacuoles; (4) shift of the Golgi apparatus toward the secreting surface; (5) changes in number, shape and direction of the mitochondria; and (6) extrusion of nuclear material into the cytoplasm. Of these, it is doubtful whether (6) is evidence of secretory activity, for it occurs in nonsecretory cells. Some of the others are inconstant and variable in different secretory cells.

It will be noted that in every case the evidence of secretory activity consists of a change in the histologic picture, and these so-called histologic evidences of secretion were established physiologically by determining that such and such histologic changes occurred simultaneously with variations in the external secretion of the gland.

It follows, then, that the proof of the secretory nature of an organ is physiologic. In fact the proof of a function is, and of necessity must be, physiologic. The most that histologic research can do is to erect a reasonable hypothesis that an organ is secretory, because its structure is similar to that of organs known to secrete.

Now the histologic picture which the pineal body presents is static. The only change observed in the pineal cells which might be interpreted as secretory is the extrusion of nuclear material into the cytoplasm. And even this has been denied, as Achúcarro, Sacristan, and Hortega believe that the material appears in the cytoplasm and is taken up *by* and included *in* the nucleus *from* the cytoplasm. In any case these nuclear

20. Bailey, Percival: Revue neurologique, 29th An., No. 6, p. 638.

21. Vincent, Swale: Secretion, Longmans, Green & Company, 1924, p. 110.

changes occur only at the time of involution of the organ, that is, after its period of supposed activity.

Moreover, the pineal body of man resembles a gland in only the grossest way. The cells are divided roughly into masses somewhat resembling acini by septums composed mainly of neuroglia and the processes of the pineal cells. Since there are no excretory ducts, a possible secretion must pass directly into the blood or lymph. But the septums contain few blood vessels compared with organs known to secrete internally, and few of the pineal cells come into contact with them (Fig. 2). Lymphatic vessels have not been demonstrated.

We can conclude only that the microscopic structure of the pineal body of man offers little support to the hypothesis that it is a secretory organ, and it follows that tumors which reproduce its structure in all likelihood do not secrete.

SUMMARY AND CONCLUSIONS

1. A clinical and pathologic report on twelve verified cases of tumors of the pineal body has been presented.

2. Of the five prepubertal cases, two showed considerable evidence of "pubertas praecox."

3. The presence of "pubertas praecox" in a patient who also shows manifestations of increased intracranial pressure and of involvement of the corpora quadrigemina is pathognomonic of pineal tumor, but there is no evidence from our histologic and pathologic studies that any secretion can come from the pineal cells to cause this syndrome.

4. The neurologic localizing signs of pineal tumors consist largely in (1) involvement of the corpora quadrigemina, i. e., oculomotor palsies, partial or complete deafness; (2) spasticity, usually bilateral; and (3) evidences of implication of the cerebellum or cerebellar tracts.

5. The pineal body is made up of two cell types: (1) neuroglia cells and (2) pineal parenchymatous cells, with a connective tissue stroma. No nerve cells are present.

6. Many of the tumors of the pineal body consist of the foregoing elements in varying proportions, and are best described as "pinealomas" of adult type.

7. Some tumors of the pineal body consist of embryonic cells of ectodermal origin, and may be called "pinealomas" of embryonic or "spongioblastic" type.[22]

22. The expenses incidental to these studies have been defrayed by the Philip Gray Fund at the disposal of Dr. Harvey Cushing for the study of brain tumors.

DISCUSSION

Dr. Frederick Tilney, New York: Dr. Bailey has produced evidence from pathology, from surgery, from anatomy and even from philosophy; why he omitted to consider the comparative side in his discussion of the pineal body, I cannot very well conceive.

I think that any one who has made an extensive comparative study of the pineal organ cannot escape the conclusion that there are forms in which it is definitely glandular.

Beginning with the lowest of the vertebrates, the cyclostomes, the epiphysis is a double structure consisting of what is called the parapineal organ and the pineal organ, both of which tend to approach the dorsal surface of the skull and there differentiate into well defined eye cups at their distal extremities. In their proximal portions near their connection with the diencephalon, their tissue is definitely glandular in character. There are well formed acini in this portion of the pineal and parapineal bodies. The same is true in the selachians, in which, however, the distal extremity of the pineal body does not differentiate as an eye; in fact, only one of the pineal organs develops to any marked extent. In all of the bony fish, the tendency for the pineal and parapineal organs to develop glandular tissue is even more pronounced. The secretory characteristics of the cells are obvious, while the glands secrete directly into the cerebrospinal fluid, which latter in these lower animals is of a different constituency from that in mammals. In amphibia, in reptiles and in birds, this same glandular evidence is easily found. All authorities who have studied this region extensively in birds have come to the conclusion that the epiphysis is definitely glandular in nature.

In mammals, the story is somewhat more difficult. The structures are a little more obscure in their histologic details, and it is less easy to discern a true glandular character in the pineal body. In mammals, only one part exists; the parapineal portion of the epiphysis disappears along with the enormous reduplications of the dorsal sac which gives the characteristic appearance to the roof plate of the interbrain in the lower vertebrates. But even in mammals, particularly during the early stages of embryonic development —and this applies not merely to the lower mammals but to those highly organized primate forms including man—it is possible to discern a stage when acinal formation is present.

I believe therefore that phylogenetically considered, this structure is a pluripotential organ, which in some of the vertebrates has become differentiated as an eye (in the cyclostomes as two eyes), with a retina, a lens and nerve fibers connecting it with the diencephalon. At the same time, the structure has developed glandular characters near its base. I also think that this same tendency has persisted even as high as man, in the early stages of whose development there is evidence of glandular formation. To consider this organ other than one of the endocrine group is, in my judgment and point of view, a mistake. I believe that in the pineal body we are dealing with a definitely endocrine gland.

Dr. Kinnier Wilson, London: Dr. Bailey's paper has been most interesting, but I wish that he had said a little more on the clinical side of the matter of pineal tumors. I should like to ask him, not in any spirit of criticism, how many of these cases were diagnosed correctly before operation or before necropsy?

From the clinical point of view, there are two symptoms to which attention might be directed as possibly of value. Neither by itself is of any specific value. When a pineal tumor is enlarged, it nearly always sets back onto the anterior colliculi, and one of the first symptoms of value in a pineal case is the appearance of paralysis of upward and downward movement of the eyes, and I have seen that in almost every case of pineal tumor.

Further, in three or four of the cases I have seen, the Argyll Robertson pupil has been present.

A second clinical point is that in these cases the patient shows a tendency to fall backward when standing in an upright position with his eyes shut. This symptom has been noted at Queen Square for many years. It is more common to have that peculiar condition when the tumor is anywhere along the anteroposterior axis of the brain than when it is situated laterally. That is an additional point which might be considered.

Our experience is that this condition of pubertas praecox is rare rather than common. I have seen only one case. In these circumstances, therefore, I can only agree with Dr. Bailey that pineal tumors remain one of the most elusive types of tumor, and that these little indications which I have given only help in making a diagnosis.

Dr. Bernard Sachs, New York: It seems to me from Dr. Tilney's anatomic study that we might possibly infer that the pineal gland may, in spite of these excellent anatomic findings, be somewhat in the position of a rudimentary organ, and I should like to ask Dr. Tilney whether there is any evidence to prove that it is still a functioning gland.

I am very much inclined to agree with Dr. S. A. K. Wilson that most of the symptoms could be satisfactorily explained by the involvement of neighboring parts.

Dr. Tilney: I think that there is evidence only in the early stages of the embryo that the tissue appears as a true aciniform structure. We have no valid evidence that it is a functioning gland in later life.

Dr. Bernard Sachs, New York: It would be difficult to assign any loss of symptoms directly to a gland that does not function.

Dr. Tilney: I was not dealing with that side of the question, but merely meant that the pineal body in one of its primary characters is glandular in nature.

Dr. Walter Timme, New York: I notice that Gilbert Horrax has collaborated in this particular presentation. I wonder whether he or Dr. Bailey will answer as to the experiments that the former carried out some eight years ago on the extirpation of the pineal body in lower forms, with resulting changes in the gonads. Those experiments were remarkable and conclusive, and I wonder whether Gilbert Horrax has retracted those views in light of this presentation, or whether he still is rather in doubt as to the results of his earlier experiments or of this particular presentation which Dr. Bailey now makes.

Dr. Percival Bailey: Answering Dr. Wilson, in most of these cases the diagnosis of tumor of the pineal body was not made prior to death, though the diagnosis was made of tumor in the region of the mid-brain and on symptoms almost similar to those which Dr. Wilson has described. An operation was not performed in many cases, because the condition was thought inoperable.

In answer to Dr. Timme, the facts which Dr. Horrax established are reported in his article. Any one may interpret these facts for himself. One should always attempt to distinguish clearly between facts established experimentally and conclusions drawn from them.

I shall answer Dr. Tilney by saying that I am familiar with his work on the pineal body, but that I cannot agree with him in the conclusions which he has drawn from it. The reasons for our disagreement will be presented in detail in the paper which will be published by Horrax and myself.

RAPIDLY DEVELOPING PARAPLEGIA ASSOCIATED WITH CARCINOMA *

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Carcinoma is always a distressing disorder. It may be latent for some time. When it develops in the stomach or prostate gland, it soon produces grave symptoms, and the distress is greatly intensified to the patient and relatives by a sudden addition of paralysis of the lower limbs when no suspicion of implication of the vertebral column or spinal cord had been entertained.

As I have not been able to read the voluminous literature, I do not know whether or not this rapidly forming paraplegia has been described in cases of carcinoma of parts of the body remote from the central nervous system, but I have observed two cases in the past few years in which this unexpected paraplegia developed without recognized warning. I did not see either patient before the paraplegia developed, but both came under my care immediately afterward.

A rapidly developing paralysis of the lower limbs or of all four limbs is suggestive of thrombosis of vessels of the spinal cord, and the resulting lesion is one of myelomalacia. It is not an infrequent development in spinal syphilis, and when it occurs in such cases it is still the result of thrombosis of the vessels of the cord sclerotic from syphilis; the resulting myelomalacia is associated with the inflammatory lesions of syphilis.

My interest in the sudden development of paralysis in syphilis of the spinal cord was first aroused by a conversation with Déjerine in 1895, when he spoke of the frequency of this occurrence, and placed it at that time at 40 per cent., as I recall, in all cases of spinal syphilis.

Thrombotic myelomalacia is likely to be sharply limited to a few segments of the spinal cord. I have reported a case with necropsy in which it was confined to the anterior half of the cord in the segments of the lower cervical region.[1] Thrombotic softening with rapidly developing paralysis may of course occur in any part of the brain stem.

An apoplectiform paraplegia in cases in which carcinoma of the stomach, prostate gland or some other part of the body has been recog-

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* From the Neuropathological Laboratory of the University of Pennsylvania and the Philadelphia General Hospital.

* Read at the fortieth anniversary of the Philadelphia Neurological Society, Nov. 29, 1924.

1. Spiller: J. Nerv. & Ment. Dis. **36**:601, 1909.

nized, is not common. Metastasis to the vertebral column is not rare. In such cases, there is usually a history of root pains of long duration, and when paralysis develops, it is usually of gradual onset. It is likely to be caused by pressure of the carcinoma on the spinal cord from an extradural tumor or tumors. There may be carcinomatosis of a large portion of the pia, or there may be carcinoma nodules on the various roots of the spinal cord, as I have observed.

I have had two cases of carcinoma in which crural paraplegia developed within an hour or so, and in one of these cases there were no previous symptoms suggesting implication of the spinal cord. In the other case, pains between the shoulders and a roentgen-ray finding of diseased vertebrae after the paraplegia developed made metastasis to the spinal column probable, but the pains before the onset of paraplegia were excusably attributed to the carcinoma of the stomach known to be present. It seemed to me that in both cases the lesion must be thrombotic, and microscopic study established this diagnosis as correct. It was necessary then to determine the manner in which thrombosis was produced, especially in the case in which previous spinal symptoms did not exist.

Although I know of no cases of sudden paraplegia associated with carcinoma of remote parts reported in the literature, as in my two cases, this rapidly developing paraplegia may in rare instances develop with tumor of the cord, as I have seen recently, when from a large tumor of the lung and abdominal cavity, possibly a hypernephroma, paralysis of the lower limbs developed within twelve hours. It may develop also with tuberculosis of the vertebral column.

Cadwalader [2] has written about the character of onset of spinal paralysis and refers to sudden onset in tumor in two cases, reported in the literature. One of these was a case reported by Foerster, who, however, gave no details. The other was reported by Müller, who observed a sudden onset of unilateral paralysis. Cadwalader has reported sudden onset of paralysis in two cases of Pott's disease, caused by myelitis from compression of the cord, in which there was microscopic evidence of thrombosis of the vessels in the spinal cord.

The two cases of tuberculous caries reported by Cadwalader are interesting in connection with my two cases of sudden paraplegia from carcinoma. In both, syphilis was denied. In Cadwalader's Case 1, the patient had been in the service of Dr. Mills. He had had abdominal pain.

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2. Cadwalader, W. B.: Character of Onset of Spinal Paralysis with Reference to Significance of Apoplectiform Type of Onset in Contrast to Slow Progressive Development of Paralysis, Arch. Neurol. & Psychiat. **6**:541, 1921; Am. J. M. Sc. **112**:546, 1911.

His right lower limb suddenly gave way while he was walking, and the weakness was associated with cramplike pain which persisted for two days, when the right lower limb became totally paralyzed. One week later, the left lower limb was similarly affected, but without pain. The paralysis in both lower limbs was then complete. Sensation was merely diminished in the lower limbs when the paralysis was first evident, but later all forms of sensation were lost in these limbs.

The necropsy revealed caries of the lower thoracic vertebrae. The lower thoracic segments, where the pressure was greatest, showed many fatty granular cells and a few lymphocytes, enlarged medullary sheaths, swollen axis cylinders and spaces from which nerve fibers had disappeared. There was moderate perivascular infiltration. The central portion of the gray matter had been destroyed and had given place to a cavity.

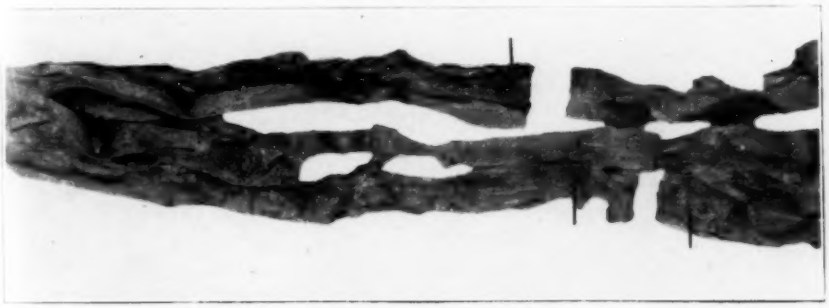

Fig. 1.—Carcinoma masses on the anterior surface of the spinal dura are indicated by lines.

These lesions were chiefly those resulting from thrombosis of blood vessels, although there were some inflammatory findings. The case resembles my Case 2, in which the patient had tuberculous instead of carcinomatous lesions of the vertebrae, and the softening was probably produced in both by occlusion of vessels of the spinal cord in passing through the diseased tissue.

Cadwalader's Case 2 was the report of a patient in my service who had been observed by me. A man had been complaining of dull aching pain just below the angle of the scapula, and two days after this pain began he had much paresthesia just below the painful areas, and on that day he suddenly lost power in both lower limbs. He had loss of touch, pain, temperature and position sensations in these limbs. He had no deformity of the vertebrae.

Necropsy showed external pachymeningitis extending from the lower cervical region to the upper lumbar region. There was a considerable

amount of necrotic cheesy material which by microscopic examination revealed the characteristic picture of tuberculous disease.

In the white substance of the upper thoracic region, there were numerous vacuoles, mononuclear cellular infiltration, slight perivascular infiltration and some fatty granular cells. In some places, the axis cylinders had disappeared; many of those remaining were swollen. The gray matter also was affected. These lesions suggested thrombosis with some inflammation. The case resembles my Case 1.

Fig. 2.—A microscopic section through one of the carcinoma masses, showing the thickness of the mass as about four times that of the dura.

Cadwalader, from his study of the literature, found that paraplegia rarely develops rapidly in tuberculous caries; when it does, there is usually a history of trauma or some undue strain on the diseased vertebral column with sudden displacement of one of the vertebrae. In neither of his two cases was there any deformity of the vertebrae at the onset of the paraplegia.

A report of my two cases of sudden paraplegia with carcinoma follows.

REPORT OF CASES

Case 1.—*History.*—P. S., a man, aged 68, who was not alcoholic and who denied syphilis, was admitted to the Philadelphia General Hospital, to my service, Nov. 21, 1919, and died Dec. 12, 1919.

The symptoms began in April, 1919, with bladder disturbance, frequency of urination, but with passage of very little urine. He was admitted at that time to the Pennsylvania Hospital, where suprapubic cystotomy was performed

by Dr. Stewart. The prostate was found to be enlarged, and it could not be removed. A drain was inserted, and the urine was drained for a week; it then passed through the wound. He remained in this hospital for two months, and the urine was still draining through the wound when he left the hospital. While at home, he used a catheter himself, as the wound healed two or three times but broke open again.

On November 11, he came home from work and lay down before supper. About one hour later, he tried to get up, but found that he could not. He felt dead from his waist down, and this loss of power and sensation continued.

On admission to the Philadelphia General Hospital, he had no voluntary motion or sensation below the level of the fifth rib on the left side and the fourth rib on the right, anteriorly and posteriorly; above these levels, motion and sensation were preserved. He said a carcinoma had been removed from his tongue. The lower limbs were completely paralyzed, but the upper limbs were not affected.

Spinal puncture showed increased pressure of the fluid, with a cell count of 3 cells to a field. The roentgen rays revealed marked osteo-arthritis from the sixth thoracic down to and including all the lumbar vertebrae.

He gradually became weaker; edema of the lungs developed; and he died, Dec. 12, 1919.

Necropsy.—The necropsy was performed by Dr. Edward Weiss. The spinal cord was softened in the upper thoracic region. When a transverse section was made through this area, it was impossible to distinguish the white from the gray matter. No note was made of any disease of the vertebrae, and evidently none was detected.

The ureteral opening in the bladder was obstructed by an enlarged prostate gland. The bladder had a thick wall, was small, and small hemorrhages were found in the upper part of its wall. The much enlarged prostate gland was very firm, and cut with greatly increased resistance. On section, whitish hyaline-like areas were seen.

I found a fibrous-like thin mass about an inch in length on the outer anterior surface of the dura at the levels of the fourth and fifth thoracic segments, which could not be separated from the dura and resembled closely the fibrous thin masses often seen on the outer surface of the dura of normal spinal cords. Other similar thin masses were found, and on microscopic examination, they were recognized as carcinoma. The mass described above was exactly at the level of the softening in the cord, and must have caused the obstruction of the circulation by implication of blood vessels.

Sections from the first thoracic segment appeared to be almost normal. Only a few holes in the lateral columns from which nerve fibers had disappeared and an occasional fatty granular cell (gitter cell) were found.

The second thoracic segment was similar, though a little more pronounced changes were observed here. Numerous similar holes were found in the posterior and lateral columns.

In the third thoracic segment, the condition was similar to that of the second thoracic segment, and the alteration was comparatively slight.

In the fourth thoracic segment, the alteration began to be pronounced at this level, but became much more intense and of the same type at the next lower level.

In the fifth thoracic segment, the changes were much greater than at any segment. The cavities from which nerve fibers had disappeared were numerous throughout the white matter. There were many fatty granular cells (gitter

cells) forming masses in certain spots, much disintegration of nerve tissue, swelling of axis cylinders, and destruction of nerve cells, but no lymphocytic infiltration. The destruction amounted to a complete transverse lesion and was a myelomalacia without inflammation.

In the sixth thoracic segment, the alteration was slight, and was similar to that of the second and third thoracic segments. It was still less at the seventh thoracic segment, where the condition of the spinal cord was almost normal.

Case 2.—*History.*—A man, aged 68, began to have pain in the abdomen in the epigastric region about Jan. 1, 1924. His condition grew steadily worse, and on February 20 he was operated on. A posterior gastrojejunostomy was performed by Dr. Gaub of Pittsburgh, as the carcinoma of the stomach was inoperable. The man recovered from the operation, and gained about 20 pounds (9 kg.). On March 28, he began roentgen-ray treatments, and came under the care of Dr. Pancoast and Dr. A. W. Phillips. He had been complaining of some pain between the shoulders for a month or so. This was attributed to the gastric carcinoma, possibly with extension. On May 30, he first noticed paralysis and numbness of the lower limbs, and rapidly lost power in them. On the afternoon of this day, he went to the toilet without assistance, but became so weak that he had to be assisted to his bed. He had increasing difficulty in moving his lower limbs during the afternoon of this day, and probably became completely paralyzed in them at that time. He had no voluntary power in them the next morning, and had to be catheterized.

I saw him first on June 5. He was completely paralyzed in his lower limbs and had no perception of pain or touch in them. The patellar reflexes were exceedingly feeble, and the Achilles reflexes were not obtained. The Babinski reflex was uncertain. The abdominal reflexes were lost. He could not pass urine, and the bowels could be moved only by enema and sometimes by the finger.

Sensations of touch, pain, heat and cold were much diminished over the trunk as high as the second intercostal space, and above this he had an area of hyperalgesia. He was much confused, and it was difficult to get him to concentrate. The upper limbs were not affected. The spontaneous pain was not severe and was in the lower part of the neck and upper part of the shoulders. The pupils were equal and of normal size.

Roentgen-ray examination of the spinal column by Dr. Pancoast showed a lesion involving the fourth and fifth thoracic vertebrae with a somewhat suspicious area in the seventh cervical vertebra. He died, June 16, 1924.

Necropsy.—Necropsy was performed by Dr. Lucké. He found carcinoma of the pylorus with metastasis to the spinal column. The bodies of the third and fourth thoracic vertebrae were softened and infiltrated with a grayish, somewhat bloody tumor mass. The growth did not press against the dura of the spinal cord, and the lesion was discovered only because of the softened condition of the vertebrae.

By microscopic examination, I found sections from the first to the fifth thoracic segments normal. They contained an unusually large number of amyloid bodies. A slight amount of degeneration was found by the Marchi method in the posterior columns at the juncture with the posterior commissure, and in the periphery of these columns.

The sixth thoracic segment showed the results of thrombosis of the vessels supplying the anterior columns of the cord. There was considerable small recent hemorrhage within these columns and numerous holes from which

nerve fibers had disappeared. At one place, a slight lymphocytic infiltration was present near the hemorrhage. The lateral columns were little affected at this level.

The seventh thoracic segment showed less implication of the anterior columns. The lateral columns were intensely altered. They presented many empty spaces from which nerve fibers had disappeared and many swollen axis cylinders, and no distinct lymphocytic infiltration. The condition was clearly one of myelomalacia and not inflammation, and was the result of vascular obstruction.

The eighth thoracic segment showed few empty spaces in the lateral columns.

The area of loss of sensation seemed to be higher than the microscopic findings could explain. The man was confused, and it was difficult to get him to concentrate. It is possible, therefore, that the loss of sensation was not quite as high as it seemed to be.

COMMENT

These two cases are illustrative of thrombosis of the spinal cord, and apoplectiform paraplegia is not likely to occur in any other condition, although thrombosis may be associated with myelitis. Myelitis sharply limited to a few segments is unusual, and it may be well to compare a case of this kind with the two already described. I have previously reported a similar case. In the three cases referred to in this paper, approximately an equal extent of the spinal cord was implicated in the lesion, but in the myelitis case the symptoms were of gradual onset and suggested tumor of the cord so strongly that operation seemed indicated to the physician in charge of the case, but was not performed. The pathologic material was sent to me for examination. I did not see the patient during his lifetime. It is difficult to explain why a myelitis should be confined to so small an area, and it is not common to find it so limited. Such lesions are likely to lead to a mistaken diagnosis of spinal cord tumor. I [3] have discussed this subject in the "Festschrift" in honor of Professor Pussep.

C. M., a man, aged 33, came under observation, Feb. 15, 1921. His chief complaint was weakness of the lower limbs. Four weeks previously, he had stopped work. A week previous to this, he noticed slight weakness in his knees associated with slight tingling. Two weeks before, he had had some pain about the waist, but this ceased within a few days.

The weakness gradually became worse, and later was associated with numbness. When he came under observation, he had partial numbness from the waist down, and his feet felt as if asleep. He could not stand alone. He had difficulty in bending his body backward and forward while sitting, and difficulty in holding the urine. He was constipated.

The patellar and Achilles reflexes were markedly exaggerated on each side, and bilateral ankle clonus and the Babinski reflex were obtained. The muscular power was fair in the right lower limb but was diminished in the left lower limb, and this limb was a little spastic. Pinprick was not recognized in either lower limb, and the loss of pain sensation extended on the trunk to the level

3. Spiller, W. G.: Folio Neuropathologica Esthoniana **3** and **4**:152, 1925.

of the umbilicus on each side. Touch, heat and cold sensations were not distinguished below the symphysis pubis and were impaired as high as the umbilicus, but the sense of position was preserved. Both lower limbs were slightly wasted. The abdominal reflexes were lost. A tumor of the spinal cord at the ninth thoracic segment was diagnosed, but the symptoms were suggestive of a lesion a segment lower than this.

The blood Wassermann test was reported as probably negative, and the spinal fluid Wassermann and colloidal gold tests were negative. The roentgen-ray report of the spinal column was negative. After the injection of air into the spinal canal for diagnostic purposes, the man became seriously ill and died within forty-eight hours, on Feb. 23, 1921.

Necropsy.—I found the eighth cervical and first thoracic segments normal, except that a very slight lymphocytic infiltration of the pia was detected. Slight meningomyelitis was found at the ninth thoracic segment, but the cord was almost normal. Intense meningomyelitis was found at the tenth thoracic segment. The vessels of the cord presented intense perivascular infiltration, with areas of disintegration of the spinal cord and swollen axis cylinders, especially in the lateral columns. At the eleventh thoracic segment, the condition was similar to that of the segment above, but was decidedly less intense. The cord was almost normal at the twelfth thoracic segment and showed only a slight lymphocytic infiltration of the pia. The findings were those of an acute lesion.

AN EXPLANATION OF THE MECHANISM OF INDUCED ROTARY AND VERTICAL NYSTAGMUS *

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A real explanation of induced rotary and vertical nystagmus should be definite as to the origins of the stimuli and the muscles stimulated. If such an explanation gives a consistent account of the phenomena, it may be accepted as dependable even though the exact neural connections are not yet demonstrated. In my proposed explanation, I shall start with the following assumption: Endolymph movement does occur, even though it consists in only a slight shift of the somewhat viscous fluid in the membranous semicircular canals; and such movement, bending the cupola and hair cells of the given crista toward or away from the canal, is the source of the stimulus to the eye muscle pairs which are known to contract in the slow or vestibular component of a nystagmus.

The endolymph movement theory of the production of nystagmus induced by rotation or douching gives a satisfactory account of horizontal nystagmus. No other theory so well explains the following facts: (1) horizontal nystagmus produced after rotation in one direction is reversed when rotation has been made in the opposite direction; (2) horizontal nystagmus produced by douching with the head 60 degrees back is reversed when the head is bent forward through 180 degrees without further douching; (3) horizontal nystagmus produced by douching with hot water is reversed when cold water is used without change of head position.

The relation of each horizontal crista to both external and internal recti is thus definite.

The crista-muscle relationship in rotary nystagmus is not so clear, and in vertical nystagmus is decidedly obscure.

PREVIOUS OPINIONS

Högyes,[1] working with rabbits, concluded that stimulation of the membranous ampullae resulted from variations in pressure in the peri-

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* Read by invitation at the Fiftieth Annual Meeting of the American Neurological Association, Philadelphia, June 6, 1924.

1. Högyes, A.: Ueber den Nerven Mechanismus der unwillkürlich associirten Augenbewegungen oder die Reflex Verbindung der zwölf Augenmuskeln mit den zwölf Ampullar Nervenden, Orvosi Hetilap., 1880, No. 17-19 (Hungarian); referred to in Jahresb. über die Fortschr. d. Anat. u. Physiol. (Literatur 1880) **9**:123, 1881.

lymph. He assigned the muscles to the cristae as follows: right labyrinth: horizontal ampulla, upper half of crista, right external rectus; horizontal ampulla, lower half of crista, left internal rectus; anterior vertical ampulla, lateral half of crista, right superior rectus; anterior vertical ampulla, medial half of crista, left inferior oblique; posterior vertical ampulla, lateral half of crista, left inferior rectus; posterior vertical ampulla, medial half of crista, left superior oblique.

Ewald,[2] working with pigeons and causing endolymph movement in isolated canals, concluded that: (1) excitation of any single canal can produce nystagmus only in a plane parallel with the plane of that canal; (2) the relation between the direction of the endolymph movement in any canal and the direction of the resulting nystagmus is definite and constant; and consequently (3) by reversal of the endolymph movement in any canal we can reverse the direction of the induced nystagmus.

Ruttin [3] assumed the anterior vertical canals to be practically in a transverse plane of the head and the posterior verticals in sagittal planes. He therefore assigned the oblique muscles to the anterior verticals and the superior and inferior recti to the posterior verticals.

Lemere [4] claimed that the anterior vertical canals were much more nearly in sagittal planes and the posterior verticals more nearly in the transverse plane. He accordingly assigned the superior and inferior recti to the anterior verticals and the obliques to the posterior verticals.

Braun and Friesner [5] said:

> If the head is tilted to one shoulder and the patient rotated, there results a vertical nystagmus. In this position, all four vertical canals are also 45 degrees to the horizontal plane. The reason we get a vertical nystagmus in this position instead of a rotary nystagmus is probably because the relative positions of the individual canals to the horizontal and the verticle planes, when the head is tilted toward the shoulder, is not the same as when the head is tilted forward.

Bard [6] said:

> Vertical nystagmus is a reflex response to an antero-posterior bending, rotary nystagmus a similar response to a lateral bending; that is why both reach their maximum sensitivity and purity when the turning which provokes them is made in the corresponding median plane.

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2. Ewald, J. R.: Physiologische Untersuchungen über das Endorgan des Nervus Octavus, Wiesbaden, 1890, p. 255.

3. Ruttin, Erich: Diseases of the Labyrinth, Trans. by H. Newhart, New York, 1914, p. 10.

4. Lemere, H. B.: Oculomotor Reaction to Labyrinth Stimulation, J. A. M. A. **71**:901 (Sept. 14) 1918.

5. Braun, A., and Friesner, I.: "The Labyrinth," New York, 1913, p. 112.

6. Bard, L.: Des conditions et du mechanisme de production des nystagmus artificiels du type rotatoire et du type vertical, Ann. de méd. **6**:175, 1919.

Jones [7] described the position of the vertical canals as follows:

The superior canal lies in a plane halfway between the frontal and sagittal; the outermost portion is placed anteriorly and the plane continues inward and backward. The posterior canal also lies in a plane halfway between the frontal and sagittal; the outermost portion however is placed posteriorly and the plane continues forward and inward.

He also said: [8]

The fibers from the horizontal canal are distributed exclusively to the external and internal recti muscles. This relation is definitely established and is very suggestive of itself that there must be different and entirely separate tracts from the different canals. . . The great difficulty has been in establishing the relation of the *vertical* canals to the plane of the nystagmus which they produce. . . Anatomically at their inmost point, they unite in a *common crus.* The movement, therefore, of endolymph in one canal cannot take place without a corresponding movement of the endolymph in the other. The plane of the resultant nystagmus, then, is the *resultant of the planes of the vertical canals.* . . With the head 120 degrees forward, on turning, the plane of the movement of the head is frontal. This frontal plane exactly bisects the planes of the two vertical canals. It will be noted that in this position the superior canal has an arc of 45 degrees inferior to this frontal plane; and that the posterior canal has an arc of 45 degrees superior to the frontal plane. It is the contribution, therefore, from these two canals that produces a reaction in the frontal plane. Clinically this is true—a rotary nystagmus is produced in the frontal plane. In a similar way, if the patient incline the head 90 degrees toward the shoulder and is turned in that position, the plane of the turning is in the sagittal plane of the head. It will be observed that the superior canal is external to the sagittal plane by an arc of 45 degrees, and that the posterior canal is also external to the sagittal plane by an arc of 45 degrees. In other words, these two canals working together produce the combined result that one canal would produce if placed in the sagittal plane. Clinically this also is true — a nystagmus is produced in the sagittal plane, a vertical nystagmus upward or downward.

A different point of view is that of Griffith,[9] a psychologist. Speaking of otologists, he says:

They have created a neurology of the central connections of the vestibular nerve that is amazing in its theoretical detail. Högyes — as an instance — has sought to differentiate twelve ampullar nerve endings and to correlate them, one by one, through the central system, with the twelve eye muscles; and Jones furnishes a still more intesting example of the fabrication of a neural mechanism suitable for the otological clinic from a few facts and a generous amount of theory.

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7. Jones, Isaac H.: Equilibrium and Vertigo, Philadelphia: J. B. Lippincott Co., 1918, p. 76.

8. Jones: Footnote 7, p. 147.

9. Griffith, Coleman R.: An Historical Survey of Vestibular Equilibration, Univ. of Ill. Bull. **20**:51 (Oct. 2) 1922. (This contains a bibliography of 1,701 titles.)

PROPOSED EXPLANATION

It is known that horizontal, rotary and vertical nystagmus, right or left and up or down as the case may be, can be induced in both eyes when only one labyrinth is functioning.[10] Suppose the right labyrinth alone is functioning. In the case of the right horizontal canal, and speaking only of the slow or vestibular component, current toward the ampulla results in contraction of the left external rectus and right internal rectus. Current away from the ampulla results in contraction of

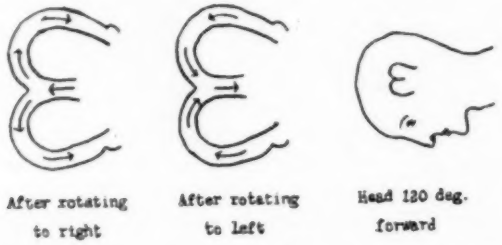

Fig. 1.—Rotary nystagmus from the right labyrinth. Currents indicated by arrows.

Fig. 2.—Vertical nystagmus from the right labyrinth. Currents predicted indicated by arrows. (Observer at patient's left and looking down on him.)

Fig. 3.—Vertical nystagmus. Two possibilities of neutrality of the common crus.

the right external rectus and left internal rectus. Thus the crista in the ampulla of the right horizontal canal controls four eye muscles. There are two other cristae in the right labyrinth, and there are eight other eye muscles. That each of these remaining cristae similarly controls four eye muscles is a tempting assumption.

In order to investigate further these right vertical canal cristae, we must be certain just how the canal currents may affect them.

10. Bárány, R.: Funktionelle Prüfung des Vestibularapparates, Verhandl. d. deutsch. Otolog. Gesellsch. Zwanzigsten Versammlung, 1911, p. 56.

After rotating to the right with the head 120 degrees forward, it is to be expected that current will flow from the common crus into the canals; after rotating to the left, current should flow from the canals into the common crus. These are the movements described by Jones and others for rotary nystagmus (Fig. 1).

After rotating to the right with the head 90 degrees to the right shoulder, it is to be expected that current will flow in the anterior vertical canal from the ampulla and in the posterior vertical canal to the ampulla; after rotating to the left, current should flow in the posterior vertical canal from the ampulla and in the anterior vertical canal to the ampulla. Any other current under these circumstances would be opposite to the direction of rotational force (Fig. 2).

But would the endolymph turn the corner opposite the upper end of the common crus? It does not matter for this explanation whether

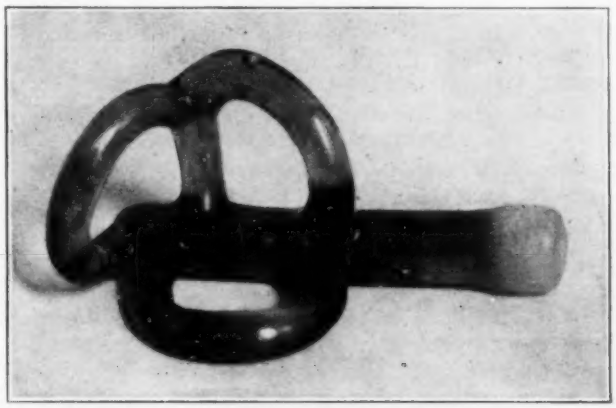

Fig. 4.—First model. When rotated this showed currents predicted.

it actually makes the turn or whether there are opposing currents on the two sides of the crus as a result of two independent whirls. The effect of stimulating one ampulla toward the canal and the other away from the canal would be the same (Fig. 3).

In an attempt to demonstrate this, a rough model was made in clay for the guidance of a glass blower. The shape and proportions of such details as ampullae and utriculus were not exact. But the three canals emptying into a common chamber, placed at proper angles, with a common crus for the verticals, were carefully molded. The glass model was made as nearly like the pattern as could be done practicably. Its canal planes are at the proper angles and its common crus is opposite a corner which is at least as sharp as that in nature (Fig. 4).

It was necessary to find some fluid which would demonstrate rotation. No clear solution would show movement, so after several

trials it was found convenient to use a mixture of one drop of indelible ink to 3½ ounces (100 c.c.) of water. This gave a suspension of particles, which took days to settle, whose movement could be easily seen against a white background. A permanently complete filling of the system was necessary, as it was found that a bubble of air at the highest point in a canal would block movement in that canal. This was accomplished by first completely filling the system and then inserting a rubber stopper through which a hypodermic needle had been passed. Such a

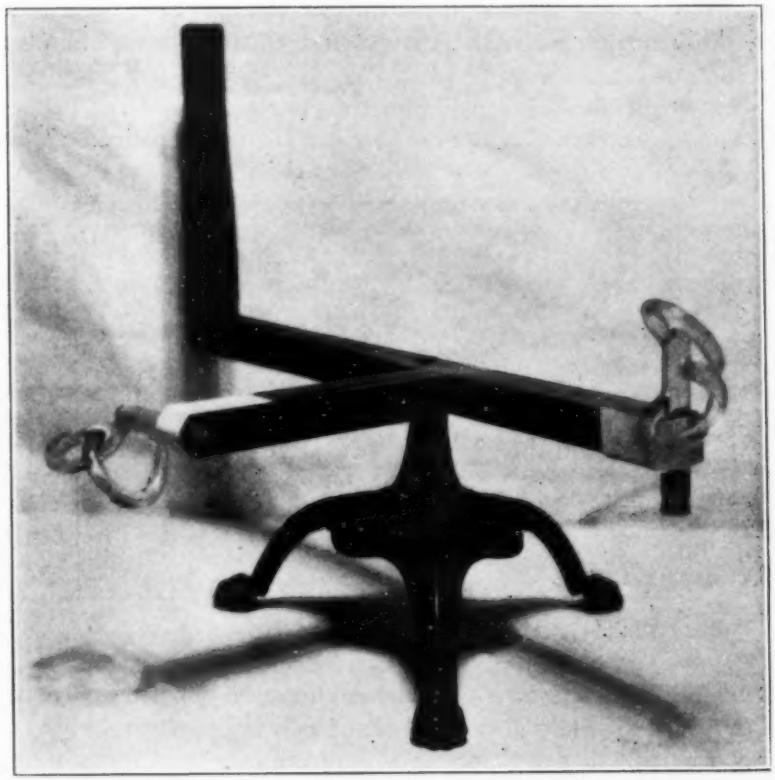

Fig. 5.—Rotating arm with two vertical canal models. At the end of the arm, model represents right vertical canals when head is forward in position for rotary nystagmus. At the side of the arm, model represents right vertical canals when head is to right shoulder in position for vertical nystagmus. After rotation, both types of current may be observed at once.

stopper could be inserted to any desired depth without endangering the glass by pressure; after this the needle was withdrawn. The filled model was then attached to one end of a rotating arm which was counterbalanced. Adhesive tape was used for holding the model in any of the three positions corresponding to those which in man give horizontal, rotary and vertical nystagmus.

In the case of each position, after even a half turn and sharp stop, rotation of fluid could be easily seen in the canal or canals thus stimulated. There was practically no movement in any canal placed perpendicular to the plane of rotation.

On rotating the verticals, placed as though the head were forward in the position for obtaining rotary nystagmus, movement was, as expected, to or from each ampulla according to the direction of turning.

On rotating the verticals, placed as though the head were on one shoulder in the position for obtaining vertical nystagmus, movement was, as predicted, definitely away from one and toward the other ampulla.

For demonstration purposes, two other models were made, but in these, for simplicity, only the vertical canals were constructed. By placing one in the position for rotary and the other in the position for

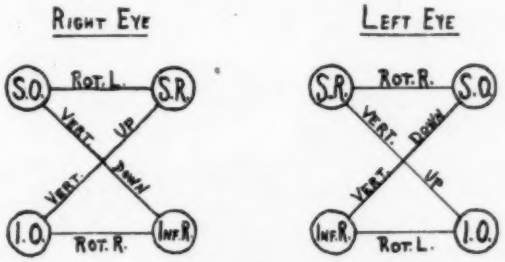

Fig. 6.—Eye muscle pairs causing rotary and vertical nystagmus.

vertical nystagmus on the same rotating arm, both types of current could be viewed simultaneously (Fig. 5).

Having, I believe, thus established the probability of such movements taking place in the human canals, it is now necessary to review the matter of muscles involved in different nystagmus movements.

Exact upward movement, when an eye is looking straight ahead, is, according to Fuchs,[11] accomplished only by the combined action of the superior rectus muscle and the inferior oblique. Similarly, downward movement is effected by the inferior rectus muscle and the superior oblique.

Rotary nystagmus, with the eye in the middle position, according to Bárány.[12] is performed as follows: inward by the superior oblique muscle and the superior rectus; outward by the inferior oblique muscle and the inferior rectus (Fig. 6).

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11. Fuchs, E.: Textbook of Ophthalmology, Trans. by A. Duane, Ed. 7, rev., Philadelphia, 1923, p. 281.

12. Bárány, R.: The Relationship Between Semicircular Canals and the Eye Muscles. The Central Mechanism in Vestibular Nystagmus, Tr. Ninth Internat. Otol. Cong., 1912, p. 593.

These are the fundamental muscle groups for such movements. Of course, when the eyes are voluntarily turned in some direction, complex conditions obtain. These need not be considered in developing the basic facts.

We may now consider how the individual muscles may be related to individual crista movements caused by canal currents. The calculation in each case follows the form of the following proposition: If $x + y$

TABLE 1.—*Calculation of Effects of Vertical Canal Currents*

RIGHT LABYRINTH

Position of Patient in Chair and Rotation Used	Canal Current Relative to Ampulla	Resulting Slow Nystagmus	Muscles Causing Slow Nystagmus	Conclusions
Head on right shoulder, rotate to right and stop	A. V. from P. V. to	Vertical up	Right S. R. Right I. O. Left S. R. Left I. O.	Right A. V. from stimulates Right S. R. Left I. O.
Head back 60 degrees, rotate to right and stop	A. V. from P. V. from	Rotary left	Right S. O. Right S. R. Left I. O. Left I. R.	
Head on left shoulder, rotate to left and stop	A. V. from P. V. to	Vertical up	Right S. R. Right I. O. Left S. R. Left I. O.	Right P. V. to stimulates Right I. O. Left S. R.
Head back 60 degrees, rotate to left and stop	A. V. to P. V. to	Rotary right	Right I. O. Right I. R. Left S. O. Left S. R.	
Head on right shoulder, rotate to left and stop	A. V. to P. V. from	Vertical down	Right I. R. Right S. O. Left I. R. Left S. O.	Right P. V. from stimulates Right S. O. Left I. R.
Head forward 120 degrees, rotate to left and stop	A. V. from P. V. from	Rotary left	Right S. O. Right S. R. Left I. O. Left I. R.	
Head on left shoulder, rotate to right and stop	A. V. to P. V. from	Vertical down	Right I. R. Right S. O. Left I. R. Left S. O.	Right A. V. to stimulates Right I. R. Left S. O.
Head forward 120 degrees, rotate to right and stop	A. V. to P. V. to	Rotary right	Right I. O. Right I. R. Left S. O. Left S. R.	

causes $a + b + c + d$, and $x + z$ causes $a + b + e + f$, it is reasonable to conclude that x causes $a + b$.

Consider only the slow (vestibular) component of after-nystagmus and that only the right labyrinth is functioning. Place the patient's head on his right shoulder when sitting in the turning chair, or lay the patient on his right side on a turning board with the right cheek on a pillow, rotate to the right and stop suddenly. This will cause endolymph movement in the *anterior vertical canal from* the ampulla, and in the posterior vertical canal toward the ampulla. Vertical nystagmus of both eyes

results with the slow component upward. This is accomplished by the *right superior rectus* muscle, the right inferior oblique, the left superior rectus and the *left inferior oblique*.

Remembering these details, consider the following. With his head back 60 degrees when sitting in the chair, rotate the patient to the right and stop. This causes endolymph movement in the *anterior vertical canal from* the ampulla, and in the posterior vertical canal from the

TABLE 2.—*Calculation of Effects of Vertical Canal Currents*

LEFT LABYRINTH

Position of Patient in Chair and Rotation Used	Canal Current Relative to Ampulla	Resulting Slow Nystagmus	Muscles Causing Slow Nystagmus	Conclusions
Head on left shoulder, rotate to left and stop	A. V. from P. V. to	Vertical up	Right S. R. Right I. O. Left S. R. Left I. O.	Left A. V. from stimulates Right I. O. Left S. R.
Head back 60 degrees, rotate to left and stop	A. V. from P. V. from	Rotary right	Right I. O. Right I. R. Left S. O. Left S. R.	
Head on right shoulder, rotate to right and stop	A. V. from P. V. to	Vertical up	Right S. R. Right I. O. Left S. R. Left I. O.	Left P. V. to stimulates Right S. R. Left I. O.
Head back 60 degrees, rotate to right and stop	A. V. to P. V. to	Rotary left	Right S. O. Right S. R. Left I. O. Left I. R.	
Head on left shoulder, rotate to right and stop	A. V. to P. V. from	Vertical down	Right I. R. Right S. O. Left I. R. Left S. O.	Left P. V. from stimulates Right I. R. Left S. O.
Head forward 120 degrees, rotate to right and stop	A. V. from P. V. from	Rotary right	Right I. O. Right I. R. Left S. O. Left S. R.	
Head on right shoulder, rotate to left and stop	A. V. to P. V. from	Vertical down	Right I. R. Right S. O. Left I. R. Left S. O.	Left A. V. to stimulates Right S. O. Left I. R.
Head forward 120 degrees, rotate to left and stop	A. V. to P. V. to	Rotary left	Right S. O. Right S. R. Left I. O. Left I. R.	

A. V., anterior vertical; P. V., posterior vertical; S. R., superior rectus; I. O., inferior oblique; S. O., superior oblique; I. R., inferior rectus.

ampulla. There results rotary nystagmus with the slow component to the left. This is accomplished by the right superior oblique muscle, the *right superior rectus*, the *left inferior oblique* and the left inferior rectus.

Compare the foregoing two pictures, noting the items common to both (italicized). So far as the endolymph movement is concerned, the one condition common to both is current in the anterior vertical canal from the ampulla. So far as muscles are concerned, we find in each the right superior rectus and the left inferior oblique. Hence, I conclude that

endolymph movement in the right anterior vertical canal from the ampulla stimulates the right superior rectus muscle and the left inferior oblique.

In a similar way, the effects of the other possible endolymph movements in each canal may be calculated.[13] The complete set of calculations is given in Tables 1 and 2.

Collecting these conclusions and adding the known data for the horizonal canals we obtain the essential facts shown in Table 3.

The common combination of horizontal and rotary nystagmus is to be expected if the head is forward or back from the exact position for obtaining pure horizontal in a given individual.

TABLE 3.—*Summary of Effects of all Canal Currents*

Canal Currents Relative to Ampullae		Muscles Stimulated	
Right anterior vertical to	or	Right inferior rectus	and
left posterior vertical from		left superior oblique	
Right anterior vertical from	or	Right superior rectus	and
left posterior vertical to		left inferior oblique	
Right posterior vertical to	or	Right inferior oblique	and
left anterior vertical from		left superior rectus	
Right posterior vertical from	or	Right superior oblique	and
left anterior vertical to		left inferior rectus	
Right horizontal to	or	Right internal rectus	and
left horizontal from		left external rectus	
Right horizontal from	or	Right external rectus	and
left horizontal to		left internal rectus	

Diagonal nystagmus is obtained after rotation with the head about 30 degrees forward and 45 degrees to one shoulder. In this position a half-strength stimulus is applied to the horizontal and vertical mechanisms. The resulting compromise is, naturally, a diagonal movement.

All the foregoing conclusions should hold good if the canals are situated approximately as described by Jones. A few degrees more or less than 45 from the sagittal plane in the case of the verticals would not change the qualitative effects.

SUMMARY

The endolymph movement theory of the production of nystagmus induced by rotation or douching is accepted as a basis for this study.

While it has given a definite explanation of crista hair movement and eye muscle action in horizontal nystagmus, no similarly definite explanation has been found for rotary or for vertical nystagmus.

A consideration of the vertical canals of one labyrinth indicates that two types of current should be expected: (1) from or to each ampulla when rotated in positions giving rotary nystagmus, as described by

13. This might be abbreviated by using the conclusions that y causes $c + d$ and that z causes $e + f$, but for clearness each calculation is made separately.

Jones and others; and (2) from one and to the other ampulla when rotated in positions giving vertical nystagmus, this being in violation of Jones' dictum that the common crus necessitates corresponding currents.

Models demonstrate these two types of current.

With these data, by comparing instances of rotary and vertical nystagmus in different conditions of production and observing common factors, conclusions are reached as to the action of each crista in each of two possible conditions of stimulation, or, in other words, the eye muscles affected by any current in any canal.

Diagonal nystagmus and combined rotary and horizontal nystagmus are explainable on this basis.

The endolymph movement theory completely and consistently accounts for the basic phenomena of induced nystagmus.

122 South Michigan Avenue.

THE CREMASTERIC REFLEX IN ITS RELATION TO THE ABDOMINAL REFLEX*

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AND
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There are many circumstances which *a priori* might lead one to regard the cremasteric reflex as an abdominal reflex at its lowest metameric level. In the first place, the contracting element, the cremasteric muscle, is justly regarded as the most caudal portion of the musculus obliquus internus abdominis, in other words, as being a portion of the muscular abdominal wall. Next, in many cases, one can from the anterior parts of its reflexogenous zone elicit a cremasteric and an abdominal reflex by the same stimulus (also the hypogastric reflex of Geigel, Dinkler, van Gehuchten and von Bechterew). Finally, these reflexes show great similarity as regards the reaction itself (a brisk, homolateral, or chiefly homolateral contraction) and the stimulus required (in most persons "stroking with a pin" is the best—often the only stimulus capable of eliciting the reflex)—both "zeitliche und örtliche Summation" being required, as Strümpell has pointed out.

Without entering further on a discussion of the literature,[1] we have tabulated the findings bearing on these two reflexes in a number of male patients treated in the neurologic clinic of the Norwegian State University (Rikshospitalet). It will be seen that the results of our investigations are to a great extent quite contrary to what might be expected from the foregoing *a priori* considerations.

As will be seen from the table, the abdominal and cremasteric reflexes show many incongruities in their behavior. In many cases, while the abdominal reflexes were brisk and of average strength, the cremasteric reflexes were feeble; in many others again, the cremasteric reflexes were of average strength or brisk while the abdominal reflexes were feeble. In eleven cases, the cremasteric reflexes were still present, while the abdominal reflexes were abolished, and in five cases the abdominal reflexes were present, while the cremasteric reflexes were absent. Only in three cases was there a possibility of this discrepancy being referable

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From the neurological clinic of the Norwegian State University (Rikshospitalet).

1. Monrad-Krohn: Om abdominalreflexerne, Kristiania, 1918.

Reflexes in a Number of Patients in the Norwegian State Hospital

	Name, Diagnosis		Epigastric and Abdominal Reflexes Right	Epigastric and Abdominal Reflexes Left	Cremasteric Reflex Right	Cremasteric Reflex Left	Periosteal Reflex of Costal Margin Right	Periosteal Reflex of Costal Margin Left	Plantar Reflex Right	Plantar Reflex Left
1.	E. N. Psychoneurosis	Abd. 1	++	++	++	++	....	....	↓↓	↓↓
		Abd. 2	++	++						
		Abd. 3	++	++						
2.	T. D. Paralysis agitans	Abd. 1	+	+	0	+	....	....	↓↓	↓↓
		Abd. 2	+	+						
		Abd. 3	0	0						
3.	H. O. P. Apoplexy	Abd. 1	++	++	++	++	++	++	0	↓
		Abd. 2	++	++						
		Abd. 3	++	++						
4.	E. A. "Etat lacunaire" (lacunar state of brain)	Abd. 1	+++	+++	++	+	++	++	0	0↓
		Abd. 2	+++	+++						
		Abd. 3	+++	+++						
5.	E. R. Tumor cerebri	Ep.	++	> ++	+++	++	++	> ++	0	↓
		Abd. 1	+++	++						
		Abd. 2	+++	++						
		Abd. 3	+++	++						
6.	J. E. S.	Abd. 1	+++	+++	++	++	+	+	↑↓	↓↓
		Abd. 2	+++	+++						
		Abd. 3	+++	+++						
7.	O. N. Acustieus tumor	Ep.	0	0	....	....	+	+		
		Abd. 1	0	+						
		Abd. 2	0	+	++	++	++	++	↓↓	↓↓
		Abd. 3	0	+						
8.	O. A. Encephalomyelopathy	Ep.	0	0						
		Abd. 1	0	0						
		Abd. 2	0	0	+	+	+	+	↑	↑
		Abd. 3	0	0						
9.	A. H. Chronic myelitis	Ep.	0	0						
		Abd. 1	0	0						
		Abd. 2	0	0	0	0	+ 0	+ 0	↑↑	↑↑
		Abd. 3	0	0						
10.	M. L. Chronic meningo-myelitis	Ep.	0	0						
		Abd. 1	0	0						
		Abd. 2	0	0	0	0	0	0	↑↑	↑↑
		Abd. 3	0	0						
11.	N. A. C. H. Dementia paralytica	Ep.	++	++						
		Abd. 1	++	++						
		Abd. 2	++	++	++	++	++	++	↓↓	↓↓
		Abd. 3	++	++						
12.	H. O. S. Dystrophia musculatorum progressiva	Ep.	++	++						
		Abd. 1	++	++						
		Abd. 2	++	++	++	++	++	++	↓↓	↓↓
		Abd. 3	++	++						
13.	T. L. Cauda equina lesion	Abd. 1	++	++						
		Abd. 2	++	++	++	++	....	....	↓	↓
		Abd. 3	++	++						
14.	A. A. Dystrophia musculorum progressiva	Abd. 1	++	++						
		Abd. 2	++	++	++	++	....	....	↓	↓↑
		Abd. 3	++	++						
15.	H. H. Tumor cerebri	Abd. 1	0	0						
		Abd. 2	0	0	++	++	....	....	↓	↓
		Abd. 3	0	0						
16.	A. O. Sclerosis multiplex	Abd. 1	++	++						
		Abd. 2	++	+	0	0	....	....	↑↑	↑↑
		Abd. 3	+	0						
17.	T. G. H. Hemianopsia	Abd. 1	++	++						
		Abd. 2	++	++	++	++	....	....	↓↓	↓↓
		Abd. 3	++	++						
18.	A. R. Arteriosclerosis cerebri	Abd. 1	++	++						
		Abd. 2	++	++	++	++	....	....	↑	0
		Abd. 3	+	—						
19.	Th. O. Tabes dorsalis	Abd. 1	++	++						
		Abd. 2	++	< ++	++	++	....	....	↓	↑↓
		Abd. 3	++	++						

Ep. indicates epigastric reflex; Abd. 1, supra-umbilical abdominal reflex; Abd. 2, umbilical abdominal reflex; Abd. 3, infra-umbilical abdominal reflex; 0, no reflex response; +, a feeble reflex response; ++, a reflex of average strength; +++, a reflex of more than average strength; ↑, extensor plantar response; ↓, flexor plantar response.

Reflexes in a Number of Patients in the Norwegian State Hospital—(Continued)

	Name, Diagnosis		Epigastric and Abdominal Reflexes Right	Epigastric and Abdominal Reflexes Left	Cremasteric Reflex Right	Cremasteric Reflex Left	Periosteal Reflex of Costal Margin Right	Periosteal Reflex of Costal Margin Left	Plantar Reflex Right	Plantar Reflex Left
20.	H. D. Hemiparalysis alterans	Abd. 1	+	++	++	++	....	....	↓↓	↓↓
		2	—	—						
		3	—	—						
21.	G. A. S. Trauma capitis	Abd. 1	++	+++	++	++	....	....	↓	↓
		2	++	++						
		3	++	++						
22.	B. J. T. General paralysis	Abd. 1	++	++	++	++	....	....	↓↑	↓↓
		2	++	++						
		3	++	++						
23.	F. A. J. Spastic paraparalysis	Abd. 1	0	0	+	+	....	....	↑↑	↑↑
		2	0	0						
		3	0	0						
24.	O. M. Poliomyelitis	Abd. 1	++	++	++	++	....	....	0	↓
		2	++	++						
		3	—	—						
25.	J. K. P. Hemiplegia dextra	Abd. 1	0	+++	+++	+++	....	....	↑	↑
		2	0	+++						
		3	0	+++						
26.	A. H. Hematomyelia	Abd. 1	0	+	....	....	....	....	↓↓	↓↓
		2	0	+						
		3	0	+						
27.	J. H. J. Dystrophia musculorum progressiva	Abd. 1	+	+	+	+	....	....	↓↓	↓↓
		2	+	+						
		3	+	+						
28.	O. G. Meningo-encephalitis	Abd. 1	++	++	0	0	....	....	↑	↑
		2	+	+						
		3	0	0						
29.	R. L. Encephalitis	Abd. 1	+	+	+ < +		....	....	↓	↓
		2	+	+						
		3	+ 0	+ 0						
30.	B. M. Sclerosis lateralis amyotrophica	Abd. 1	0	0	+	+	....	....	↓↓	↓↓
		2	0	0						
		3	0	0						
31.	M. S. Neuritis multiplex	Ep.	0	0	0	0	....	....	↓↑	↓0
		Abd. 1	0	0						
		2	0	0						
		3	0	0						
32.	E. F. Sclerosis multiplex	Abd. 1	+	0	0	+	+	++	↑↑	↑↑
		2	0	0						
		3	0	+						
33.	S. R. (pol) Neuritis retro bulbaris	Abd. 1	0	0	+	+	++	++	↓	↓
		2	0	0						
		3	0	0						
34.	R. H. Dementia paralytica	Ep.	++	++	++	++	++	++	↓↓	↓↓
		Abd. 1	++	++						
		2	++	++						
		3	++	++						
35.	A. B. Jacksonian epilepsy (tumor cerebri)	Ep.	++	+	++	++	++	+	0	↑
		Abd. 1	++	+						
		2	++	+						
		3	++	+						
36.	G. O. Hemianopsia	Abd. 1	+	++	++	++	....	....	↓↓	↓↓
		2	+	++						
		3	+	++						
37.	Hj. E. Tumor cerebri	Ep.	+	++	++	++	+	++	0	↑↑
		Abd. 1	+	++						
		2	+	++						
		3	+	++						
38.	H. S. Epilepsy	Ep.	(+)+	++	+	++	++	++	↓↓	↓↓
		Abd. 1	++	++						
		2	++	++						
		3	++	++						
39.	O. M. Epilepsy	Ep.	++ <	++	++	++	++ < ++		↓↓	↓↓
		Abd. 1	++ <	++						
		2	++ <	++						
		3	++ <	++						

Reflexes in a Number of Patients in the Norwegian State Hospital—(Continued)

	Name, Diagnosis		Epigastric and Abdominal Reflexes Right	Left	Cremasteric Reflex Right	Left	Periosteal Reflex of Costal Margin Right	Left	Plantar Reflex Right	Left
40.	S. J. Dementia	Ep.	+	+						
		Abd. 1	+	+						
		Abd. 2	+	+	+	+	++	++	↓↓	↓↓
		Abd. 3	+	+						
41.	A. P. Poliomyelitis ant. ac.	Ep.	+	0						
		Abd. 1	+	0						
		Abd. 2	+	0	+	+	+	+	↓↓	↓↓
		Abd. 3	+	0						
42.	R. R. Tumor cerebri	Ep.	+	—						
		Abd. 1	++	+						
		Abd. 2	++	+	++	++	++	++	0↓	↓↓
		Abd. 3	++	+						
43.	O. L. F. Spastic paraparalysis	Ep.	?	?						
		Abd. 1	0	0						
		Abd. 2	++	> ++	+	+	++	++	↑↑	↑↑
		Abd. 3	+ ?	+ ?						
44.	E. R. Vertigo	Ep.	++	++						
		Abd. 1	++	++						
		Abd. 2	++	++	++	++	....	....	↓↓	↓↓
		Abd. 3	++	++						
45.	A. H. R. Poliomyelitis ant. ac.	Abd. 1	0	0						
		Abd. 2	0	0	++	++	....	....	↓↓	↓↓
		Abd. 3	0	0						
46.	H. E. Brown-Séquard syndrome	Ep.	+	0						
		Abd. 1	0	+						
		Abd. 2	0	+	0	+	....	....	↑	↓
		Abd. 3	0	+						
47.	O. S. Organic brain disease	Abd. 1	+	+						
		Abd. 2	+	+	+	+	....	....	↓	↓
		Abd. 3	+	+						
48.	J. A. Astasia-abasia	Ep.	++	++						
		Abd. 1	++	++						
		Abd. 2	++	++	++	++	....	....	↓↓	↓↓
		Abd. 3	++	++						
49.	O. S. Tabes dorsalis	Ep.	++	++						
		Abd. 1	++	++						
		Abd. 2	++	++	+	+	0	0	↓	↓
		Abd. 3	++	++						
50.	Th. J. Poliomyelitis ant. ac.	Ep.	++	++						
		Abd. 1	++	++						
		Abd. 2	++	++	++	++	++	++	↓↓	↓↓
		Abd. 3	++	++						
51.	O. G. Sclerosis multiplex	Ep.	0	0						
		Abd. 1	0	0						
		Abd. 2	0	0	0	0	+	+	↑↑	↑↑
		Abd. 3	0	0						
52.	A. F. Spastic paraparalysis	Ep.	0	0						
		Abd. 1	0	0						
		Abd. 2	0	0	+	+	++	> ++	↑↑	↑↑
		Abd. 3	0	0						
53.	P. H. Aphasia	Ep.	+ 0	+						
		Abd. 1	+ 0	++						
		Abd. 2	+ 0	++	+	+	0	0	0	↓
		Abd. 3	+ 0	++						
54.	O. S. Poliomyelitis ant. ac.	Ep.	+	+						
		Abd. 1	+	+						
		Abd. 2	+	+	0	+	+	+	↑	↓↓
		Abd. 3	0	0						
55.	K. M. M. Sclerosis multiplex	Abd. 1	0	0						
		Abd. 2	+	+	++	> ++	....	....	↑↑	↑↑
		Abd. 3	+	+						
56.	J. H. Meningomyelitis	Abd. 1	0	0						
		Abd. 2	0	0	0	0	....	....	↓	↓
		Abd. 3	0	0						
57.	A. H. Poliomyelitis acut. ant. recidivans	Abd. 1	++	++						
		Abd. 2	++	++	++	++	....	....	0	↑
		Abd. 3	++	++						

Reflexes in a Number of Patients in the Norwegian State Hospital—(Continued)

	Name, Diagnosis		Epigastric and Abdominal Reflexes Right	Epigastric and Abdominal Reflexes Left	Cremasteric Reflex Right	Cremasteric Reflex Left	Periosteal Reflex of Costal Margin Right	Periosteal Reflex of Costal Margin Left	Plantar Reflex Right	Plantar Reflex Left
58.	N. F.	Ep.	++	++						
	Atrophia nervi optici	Abd. 1	++	++	++	++	....	....	↓↓	↓↓
		2	++	++						
		3	++	++						
59.	O. S.	Ep.	+	+						
	Cerebrospinal syphilis	Abd. 1	+	+	0	0	....	....	↑↑	↑↑
		2	+	+						
		3	+	+						
60.	R. H. H.	Ep.	++	++						
	Poliomyelitis acuta anterior	Abd. 1	++	++	++	++	....	....	↓↓	↓↓
		2	++	++						
		3	++	++						
61.	J. T.	Ep.	0	0						
	Apoplexia recidivans	Abd. 1	0	0	0	0	....	....	↓	↓
		2	0	0						
		3	0	0						
62.	O. S.	Ep.	++	++						
	Paralysis of the oculomotor nerve	Abd. 1	++	++	+	0	....	....	↓↓	↓↓
		2	++	++						
		3	+	+						
63.	A. N. T.	Ep.	0	0						
	Myelitis chron.	Abd. 1	0	0	0	0	....	....	0↓	↑
		2	0	0						
		3	0	0						
64.	H. K. S.	Ep.	0	0						
	Myelitis acuta	Abd. 1	0	0	0	0	....	....	0	0
		2	0	0						
		3	0	0						
65.	O. B.	Ep.	0	0						
	Sclerosis multiplex	Abd. 1	0	0	0	0	....	....	↓↑	↓↑
		2	0	0						
		3	0	0						
66.	P. H. H.	Ep.	+	0						
	Sclerosis lateralis amyotrophica	Abd. 1	+	0	++	++	....	....	↑	↑
		2	+	0						
		3	+	0						
67.	R. O.	Ep.	++	++						
	Dystrophia adiposo genitalis	Abd. 1	++	++	+	+	....	....	↓↓	↓↓
		2	++	++						
		3	++	++						
68.	F. T.	Ep.	0	0						
	Arteriosclerosis	1	0	0						
		2	0	0	+	+	. ..	....	↓0	0↓
		3	0	0						
69.	J. T.	Ep.	+	+						
	Hemiplegia (syphilis)	Abd. 1	+	+	+	< +	....	....	↑	↓↑
		2	+	+						
		3	+	+						
70.	R. H. T.	Ep.	++	++						
	Epidemic encephalitis	Abd. 1	+++	+++	++	++	....	....	↓↓	↓↓
		2	+++	+++						
		3	+++	+++						
71.	M. H.	Ep.	++	++						
	Spondylitis cervicalis	Abd. 1	++	++	++	++	....	....	0	0
		2	++	∧∧ ++						
		3	++	∧∧ ++						
72.	H. E.	Ep.	+	∧∧ +						
	Hemiparalysis et hemihyperesthesia	Abd. 1	+	∧∧ +	++	++	....	....	↑	↓
		2	+	∧∧ +						
		3	0	0						
73.	A. J.	Ep.	++	++						
	Arteriosclerosis (syphilis)	Abd. 1	++	++	+	+	....	....	↓↓	↓↓
		2	++	++						
		3	++	++						
74.	H. G. B.	Ep.	+	+						
	Glioma cerebri	Abd. 1	+	+	+	+	....	....	↑	↑
		2	+	+						
		3	+	+						
75.	M. A.	Ep.	++	++						
	Sclerosis multiplex	Abd. 1	++	++	++	++	....	....	↑	↑
		2	++	++						
		3	++	++						
76.	W. W.	Ep.	+	+						
	Neurosis	Abd. 1	++	++	++	++	....	....	↓	↓
		2	++	++						
		3	++	++						
77.	E. O. H.	Ep.	0	0						
	Tabes dorsalis	Abd. 1	++	++	++	++	....	....	↓	↓
		2	++	++						
		3	++	++						

Reflexes in a Number of Patients in the Norwegian State Hospital—(Continued)

	Name, Diagnosis		Epigastric and Abdominal Reflexes Right	Epigastric and Abdominal Reflexes Left	Cremasteric Reflex Right	Cremasteric Reflex Left	Periosteal Reflex of Costal Margin Right	Periosteal Reflex of Costal Margin Left	Plantar Reflex Right	Plantar Reflex Left
78.	G. A. Tabes dorsalis	Ep.	++	++						
		Abd. 1	++	++						
		Abd. 2	++	++	++	++	....	....	↓↓	↓↓
		Abd. 3	++	++						
79.	A. W. Epidemic encephalitis	Ep.	++	++						
		Abd. 1	++	++						
		Abd. 2	++	++	++	++	....	....	↓↓	↓↓
		Abd. 3	0	0						
80.	P. B. F. Paresis brachialis dext.	Ep.	++	++						
		Abd. 1	++	++						
		Abd. 2	++	++	++	++	....	....	↓↓	↓↓
		Abd. 3	++	++						
81.	G. W. Sclerosis multiplex	Ep.	0	0						
		Abd. 1	0	0						
		Abd. 2	0	0	+	+	....	....	↑	↑
		Abd. 3	0	0						
82.	S. H. Epidemic encephalitis	Ep.	++	++						
		Abd. 1	++	++						
		Abd. 2	++	++	++	++	....	....	↑↓	↓↑
		Abd. 3	++	++						
83.	A. E. H. Sclerosis multiplex	Ep.	0	0						
		1	0	0						
		2	0	0	0	0	....	....	↑↑	↑↑
		3	0	0						
84.	P. M. P. Syringomyelia	Ep.	++	++						
		Abd. 1	++	++						
		Abd. 2	++	++	++	++	....	....	↓0	↓0
		Abd. 3	++	++						
85.	J. S. Epilepsy	Ep.	++	++						
		1	++	++						
		2	++	++	++	++	....	....	↓↓	↓↓
		3	++	++						
86.	E. G. Tumor medullae spinalis	Ep.	++	++						
		Abd. 1	++	++						
		Abd. 2	++	++	0	0	....	....	↓↓	↓↑
		Abd. 3	++	++						
87.	G. L. Cerebrospinal syphilis	Ep.	+	+						
		Abd. 1	+	+						
		Abd. 2	+	+	++	++	....	....	↓↑	↓↑
		Abd. 3	+	+						
88.	R. S. Dementia paralytica	Ep.	++	++						
		Abd. 1	++	++						
		Abd. 2	++	++	++	++	....	....	↓	↓
		Abd. 3	++	++						
89.	Th. S. Meningoencephalitis	Ep.	+	+						
		Abd. 1	+	+						
		Abd. 2	+	+	++	++	....	....	↓	↓
		Abd. 3	+	+						
90.	S. H. O. Tumor cerebri	Abd. 1	++	0						
		Abd. 2	++	0	0	0	++	++	↓↑	0↑
		Abd. 3	++	0						
91.	K. G. Sclerosis multiplex	Abd. 1	0	0						
		Abd. 2	+	0	+	+	+	+	↓↑	↓0
		Abd. 3	0	0						
92.	J. J. Cerebrospinal syphilis	Ep.	++	+						
		Abd. 1	++	+						
		Abd. 2	++	+	++	+	....	....	0	↑
		Abd. 3	++	+						
93.	O. G. B. Epilepsy	Abd. 1	++	++						
		Abd. 2	++	++	++	++	++	++	↓↓	↓↓
		Abd. 3	++	++						
94.	I. B. Atrophia n. optici	Abd. 1	0	0						
		Abd. 2	+	0	++	++	++	++	↓↓	↓↓
		Abd. 3	+	++						
95.	H. P. Morbus nullus	Abd. 1	++	++						
		Abd. 2	++	++	+	+	++	++	↓	↓
		Abd. 3	++	++						
96.	M. F. Sclerosis multiplex	Abd. 1	++	++						
		Abd. 2	++	++	++	++	++	++	↓0	↑0
		Abd. 3	++	++						

to different segments of the cord having been unequally affected. Clinical experience therefore does not seem to confirm the *a priori* supposition mentioned in the foregoing that the cremasteric reflex can be regarded as a lower abdominal reflex. It seems reasonable to assume that the arc of the cremasteric reflex is different from that of the abdominal reflex (probably a shorter and therefore a less exposed arc).

The relation of the abdominal and cremasteric reflexes to the plantar reflexes and in some of the cases to the periosteal reflex of the costal margin is also made clear in the table.

THE PHYSIOLOGY OF THE FEEBLEMINDED*

GEORGE S. STEVENSON, M.D.
MINNEAPOLIS

In a previous report,[1] attention was called to some circulatory differences between two groups of feebleminded persons. Other more or less detailed studies have been carried on which were specifically concerned with roentgenographic observations of visceral form and activity and of food intake.

ROENTGEN-RAY BIOPSIES[2]

Ludlum[3] has found that in certain cases of mental backwardness internal anomalies of development and function exist. It was felt desirable in a survey of this sort to investigate as far as possible by the use of roentgenography. While the original plan was more of the nature of an extensive biopsy, and this was carried out on some cases, it was finally decided to concentrate on one functional system at a time. The first work was done with the heart; later work, with the gastro-intestinal tract.

HEART ROENTGENOGRAPHY

As an accessory means of studying the circulatory apparatus, and as part of a general biopsy, teleoroentgenograms and orthodiagrams were made of the hearts of a number of defective boys, using high grade high-school students as controls. The limitations of the method and the lack of results caused this study to be abandoned. The results conformed to a degree to the principle that stature has much to do with the size and shape of the shadow, the short stout person having the prominent (transverse) heart and the thin one the hidden (vertical heart). The rating of the relations has not been sufficiently worked out to make it applicable to this study. The hearts in these boys were not pathologic in the sense of the ordinary physical examination. They did not even show a marked functional disturbance. The anatomy of the

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1. Stevenson, G. S.: Cardiovascular Physiology of the Mental Defective, Arch. Neurol. & Psychiat. **12**:507 (Nov.) 1924. Paragraph 1, last sentence, substitute "between" for "on" before the word "conditions."

2. These observations were made by Dr. A. E. Colcher, and the details may be made the subject of a further report.

3. Jatho, Edna R., and Ludlum, S. D.: A Study of the Internal Stigmas of Degeneration in Relation to Metabolism and Disturbance of the Cerebral Cortex in Children, Arch. Neurol. & Psychiat. **7**:167 (Feb.) 1922.

hearts would be expected to show only slight, if any, deviation from the normal. The method is not refined enough for this, considering the difficulties presented by markedly pathologic hearts.

GASTRO-INTESTINAL ROENTGENOGRAPHY

Owing to the absence of definite leads for gastro-intestinal work, a survey of the tract, as done in clinical work, was planned. This was first carried out on the same selected group used for cardiac studies and drug reactions, boys who, as far as possible, deviated from the normal in intelligence only. Examination of these was practically negative.

TABLE 1.—*Roentgenographic Findings in the Gastro-Intestinal Tract*

Condition	Cases	Condition	Cases
Stomach		**Colon (as a whole)**	
Large	5	Narrow	2
Small	1	Much gas	5
Displaced to right	2	Large	6
Low	5	Sluggish	2
Narrow pylorus	1		
Barium retention after 6 hours	6	**Sigmoid**	
Sluggish motility	9	Large	3
		Twisted	1
Duodenum		Displaced	1
Deformed cap	1		
Slowly emptying cap	5	**Rectum**	
Delay in second and third part	3	Large	4
Cecum		**Expulsion Time**	
Large	2	24	1
Ileocecal incompetency	7	48	4
		72	8
Ascending Colon		96	7
Doubling	3	120	1
Large	1	Over 120 (144, 192, 216)	3
Transverse Colon		**Carmin, 10 cases**	
Low	10	Appearance over 24 hours	4
		Elimination over 48 hours	4
Descending Colon			
Hypotonic	1		
Misshapen	2		
Large	1		

Following this, a group of persons was selected who were not only intellectually handicapped, but who also for some reason were doing poor school work for the mentality they had. Later, some were selected who did not show this peculiarity, but the majority were low in efficiency as well as in mentality. There were twenty-four in all.

The method consisted of routine preparation: limitation of food, barium meal at 9 a. m. and at the time of examination at 3 p. m., subsequent observations to follow the meal until its disappearance. Later a barium enema was administered and observations were made. Finally, in ten cases, carmin was given and the time of appearance in and of disappearance from the stool noted.

No particular anomalies or pathologic conditions were sought, but all seen during the examinations were noted. Table 1 gives briefly the results of these examinations.

This table shows no regularity in the abnormalities present. No one condition is present in half the cases. This probably represents the distribution of pathologic conditions in the feebleminded. The possible significance will be discussed later.

CONCLUSION

The feebleminded show many anomalies of form and function of the gastro-intestinal tract with no characteristic single anomaly.

FOOD INTAKE IN FEEBLEMINDED CHILDREN

Many of those who care for the feebleminded have had the impression that the feebleminded require more food than normal persons. Whether this, if a fact, represents a physiologic peculiarity or merely a lack of restraint on the appetite, is questionable. The latter has been generally assumed to be the case. Letters directed to several institutions asking the question brought out varied replies. Of the six replies received, two unqualifiedly stated that the feebleminded ate more; two stated that they ate more due to freedom from worry and from refinement. One said that the lower grades of feebleminded ate more, while one said that some had large and some had small appetites, but, on the whole, they would range about as those of normal persons.

The first question taken up is whether a high intake really means a high requirement. Experience has shown that in institutions there is always a group, fairly large in size, marked as heavy eaters. Four boys were selected from this group on the basis of uniformity in age and conformity to normal persons in height and weight. They were allowed to go about their usual work without restraint on their activities. They were instructed to receive no food beyond that which was given to them at meal times, and persons who came into contact with them were instructed to give them nothing. Gifts of candy were turned in to be weighed before being eaten. For ten days, the weight and kind of unrestricted voluntary intake of food was recorded. At the end of this period, a computation of the caloric values showed that each of these four boys consumed practically 4,000 calories daily. Since these boys were selected from a gluttonous group, this figure is much too high for the average child in the institution.

In order to determine whether this excessive intake in this type of boy was really necessary, it was decided to begin at 4,000 calories per day, reduce the diet 200 calories every third day and take the weights of the boys daily. A consistent loss of weight on a reduced diet would indicate its insufficiency—other influences being absent. Two of the children cooperated during this period of reduction, the other two, G. M. and J. F., were known to receive food other than that given them under the conditions of the experiment.

During reduction by this method to 2,600 calories, there was a definite though slight loss of weight in three of the boys. The terminal weight was lower, and a comparison of the average weights during the separate thirds of this period showed a more or less consistent fall. The loss of weight was not marked, but amounted to about 1 kilogram.

Having reached a level of 2,600 calories, the diet was maintained at this value for ten days. During these ten days there were fluctuations in weight, but the terminal weights averaged no lower than those of the middle or first third of the ten day period.

CONCLUSION

While reduction of diet occasioned a slight loss of weight, adjustment occurred rapidly, and the boy was able to go ahead on the reduced diet without loss of weight when further reduction was stopped.

TABLE 2.—*Food Intake in a Group of Gluttons*

Average Observations	G. M., Aged 19, Height 179.5 Cm., Lightest Farm Work		J. F., Aged 16, Height 166 Cm., School Work		E. S., Aged 19, Height 172 Cm., On Grocery Wagon		J. B., Aged 18, Height 170 Cm., Clearing Lawns	
	Calories	Weight	Calories	Weight	Calories	Weight	Calories	Weight
10 day period	3,955	64.8	3,984	54.4	3,899	50.6	4,007	62.0
1st 3 day period	4,034	64.8	4,090	55.0	4,041	50.5	4,011	61.9
2d 3 day period	3,827	64.9	3,538	56.0	3,585	51.4	3,727	62.7
3d 3 day period	3,601	64.1	3,623	55.4	3,597	51.1	3,703	62.0
4th 3 day period	3,394	64.6	3,415	55.0	3,403	50.9	3,403	61.4
5th 3 day period	3,157	63.8	3,306	55.4	3,077	50.6	3,217	60.9
6th 3 day period	2,994	63.5	2,996	56.1	3,015	50.5	3,998	60.8
7th 3 day period	2,793	63.6	2,864	55.6	2,882	50.5	2,800	60.6
8th 3 day period	2,568	63.7	2,587	55.9	2,600	50.2	2,595	60.3

It cannot be assumed from this that we should be able to reduce the total intake of the children in the proportion of 4,000 to 2,600 calories, because we have selected the gluttons. However, if this apparent fact of excessive intake can be confirmed, it would indicate that, as a matter of economy, boys of this type could be segregated and fed separately. Foods of bulk could be used in place of those having high caloric value. The desires of the child have, of course, to be considered. A study of the likes and dislikes of the child showed that it was a simple matter to bring about the reduction mentioned. In fact, at the 2,600 level the boys were better satisfied than at about 3,200, because their likes and dislikes were better known by that time.

GENERAL CONCLUSIONS

No individual line of investigation has yielded a clue to what feeblemindedness is. No finding has been consistent enough to be considered the rule. One thing, however, is evident; there are many peculiarities of varied sorts to be found in the physiology of the feebleminded. No set or symptom is common to all or even to a large majority. Each

case has its peculiar set. This inconsistency in type, with marked consistency in the presence of defects is characteristic. Our views of the importance of one or other of the defects mentioned may be and probably will be modified, but the fact remains that defects do exist and exist in an unusual degree. There is but one possible conclusion. Feeblemindedness is no entity; it must be conceived as a disease of the deficiency group. It probably has a varied etiology. Possibly certain factors are common in the etiology of many types of cases—social influences especially. It is a point of note in this connection that many of these defects are handicaps to economic success in normal people.

COMMENT

It has been stated previously that the social criterion is biologically unsound. This is meant to apply to the individual, and it does not imply that the influence of society can be disregarded. The line of economic sufficiency is arbitrary from the physiologic point of view. Society is an important factor in our problem, since it determines the degree of handicap necessary for economic failure. It tends to exclude from the ranks of independent society those whose summed up handicaps extend beyond a certain maximum, and allows them social recognition directly in proportion to their abilities. Thus the handicaps come to be determiners of social position. Below the level of complete independence we come to have what Austin Freeman [4] terms the subman, whose difference "from the completely normal and completely fit . . . is simply a matter of deficiency . . . . They are inferior human beings, defective mentally, morally and often physically . . . . They are probably inferior to members of any aboriginal race known to us." While the resemblances to primitive races have been freely emphasized, the differences, always in favor of primitive man, have not escaped notice. Above all, primitive man is economically independent, and is not physically defective. What defects he has, judged from civilized standards, are *balanced by qualities* which he as a race has not yet discarded for something better. But the inferior civilized man is not economically independent. He is the lower end of a selection tendency which extends in both upward and downward direction from normal. He has fallen below the requirement for social sufficiency and has not the *balancing qualities* even to make him successful in a primitive society; he is often out of work, is relatively unskilled as is no other persisting, living thing, and is probably a biologic regression not in one line but many—mainly mentally but also anatomically and physiologically. As in his gravitation to the lower level, the individual

4. Freeman, Austin: The Sub-Man, Eugenics Rev., July, 1923.

carries his handicap with him, leaving it there often with his progeny, the lower in the scale we search for these peculiarities or stigmas, the more of them we find. This is the result of a civilization which has so abundantly equipped the fit that the overflow has made possible the survival of the otherwise dependent, who in primitive states would have been eliminated.

It cannot be assumed that all socially gravitating defects have a bearing on the production of the feebleminded syndromes. It makes a great difference whether the handicap is accidental or innate. The result of the accidental handicap would be an elevation of the social level. Such should be disregarded in the present discussion. The innate handicap, whether it be a natural variation, a regression or something inherited, has the possibility of being transmitted. This regression may not be anything acute or sudden. There is a regressive as well as progressive tendency constantly in action, for the reason that no offspring is exactly like the parent. Some regressive tendencies are compensated for by other progressive ones in the same person, with a more or less stationary result. In other cases, an approach to economic decline takes place.

Marriage is most apt to occur within the same social caste. The effect of this at the level where economic sufficiency is on the verge of collapse is evident. The determining handicaps of all sorts, mental, physical and physiologic, will tend to be concentrated in a part of the next generation. Always something lower is produced.

Probably from his very nature as compared with other mammals, man's success is closely related to his various mental qualities. Defects in these qualities, whether general or limited, make for social gravitation. Consequently, mental inferiority is the most prevalent defect of the lower social levels. On the borderline, where the summation of functional defects has been more concentric with less tendency toward unbalance, there results a person who may, under favorable conditions, be able to meet the requirements for economic independence more or less adequately. The group with mental ages from 8 to 12 years contains many persons who show this.

Thus the possibility of concentration of handicaps at the lower level brings these handicaps most frequently into combinations of which mental defect is an important part. It produces a syndrome of mental inferiority with anomalies of anatomy and function. As the stock persists at the sub-sufficient level, the more fixed, the more numerous, and, in some cases, the more balanced, do these inferior qualities become.

Two common defects may be compared in the light of this concept to show their effects as economic handicaps; their tendency to social gravitation and their concentration at the sub-sufficient level. In the

case of defective hearing, the handicap might be classed as less important than defective vision. However, society has evolved an efficient neutralizer of the economic effects of defective vision which it has not done for hearing defects. It is a statistical fact [5] that hearing correlates significantly and highly with mental insufficiency or inferiority, and that vision has no significant correlation. Similarly, psychopathic tendencies are more prevalent among the feebleminded, and the ancestry of feebleminded persons is often loaded with psychopathy, with or without serious intellectual defect.

The process of gravitation has an important evolutionary value. It is the process whereby progressive civilization purges itself of its many and varied undesirable characteristics. The tendency, however, has been to permit a stasis to develop in the lower level of society. He who wishes to restrain this process of elimination might accordingly excise his own bladder and bowel and expect only eternal bliss to result. Evolving man must have this outlet. The causes for this stasis have already been reviewed; the fact of the segregation of social classes has no doubt obscured the presence of the problem. Possibly the personal feeling of inferiority prevalent among those of the higher social levels has interfered with freedom of action in assisting nature in the elimination of these qualities. Certainly with the present differential birth rate, nature cannot perform the task alone. One has only to look at the mentally defective, psychopathic, weedy, undersized, diseased, deformed, maldeveloped, malfunctioning individuals who are dependent in endless ways on society for unearned sustenance, to get an incentive to think.

University of Minnesota.

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5. Mallory, J. N.: A Study of the Relation of Some Physical Defects to Achievement in the Elementary School, George Peabody College for Teachers, 1922.

Abstracts from Current Literature

A Case of Porobulbia and Solitary Central Neurinonia (with a Contribution to the Question of the Infundibular Process). H. Joséphy, Ztschr. f. d. ges. Neurol. u. Psychiat. **39**:62 (Sept.) 1924.

The author reports a case of a German woman, aged 22, who was somewhat feebleminded in childhood. Her left leg was paralyzed, but she had never had any convulsions. At the age of 16 years and 6 months, she began to show signs of a serious disorder. She often stared straight ahead, and was often preoccupied and confused. She began to have attacks of vertigo, fell down, could not rise again and was unconscious after her falls. She developed a paralysis of the entire left side, preceded by a paralysis of the left leg, and gradually lost her sight. Her menses ceased with the beginning of this illness. On examination at this time, she weighed 40 kg. She appeared younger than her age indicated. On her breasts and arms were brownish-yellow discolorations ("liver spots"). The left pupil was larger than the right; both did not react to light, and both fundi showed swelling of the papillae. There was a left facial palsy. The tendon reflexes were hyperactive on the left side, the muscular power was decreased on the left, the left leg was dragged in walking, and the patient reeled to the left side. There was disturbance in the bladder function. The patient was definitely defective mentally. The Wassermann reaction in the blood and spinal fluid was negative, and the spinal fluid pressure was increased.

Soon after the onset of her illness, the patient became sleepy — completely apathetic and had to be fed. Sight decreased more and more, and the signs of a left-sided hemiplegia were more apparent. About two months after her illness set in, a left-sided Babinski sign and an ankle clonus could be obtained. The swelling of the papillae receded, and secondary optic atrophy set in. The condition of lethargy persisted. About nine months after onset, the patient had an epileptiform attack. About eight days later, her condition improved; she became more active, spoke spontaneously, sang, laughed and exchanged witticisms. This condition lasted for one year, the menses reappeared, and a gradual gain in weight was apparent. The disturbance in the reflexes disappeared, although there remained a slight increase in the reflexes on the left and a rigidity of the left arm. At the end of a year, the patient again became somnolent, vomited frequently and had numerous epileptiform attacks. She lost 6 kg. in weight. Her condition again cleared up, however, after four months, and a puncture of the right lateral ventricle was performed, without revealing any signs of a tumor. Her condition was good at this time. Her weight was 70 kg.; she had no differences in her tendon reflexes, and the muscle power was the same on both sides. She reached a weight of 79 kg. about three years after the onset of her illness, a gain of 39 kg. The mammae and abdomen were very fat. She began to have a definite polyuria, and attacks of vertigo, and epileptic convulsions set in. She again lost weight rapidly, however, dropping to 50 kg. four years after the onset of her trouble. Her menses ceased again at this time. She died suddenly in collapse several months later.

The patient showed two types of symptoms — neurologic and endocrine. Neurologically, she showed a spastic, left-sided paralysis, and later an optic

neuritis which quickly led to atrophy and complete amaurosis. Soon after her illness, she became apathetic, and her reflexes became hyperactive. After several months, all this cleared up, but a year later returned, this time with vomiting and several epileptiform attacks. This condition lasted three months, then suddenly disappeared. About one and one-half years after this, she again suddenly became ill, and quickly died.

In the endocrine sphere, we find a cessation of menses at the beginning of the illness, although they had always been regular until that time. When the first period of somnolence set in, a marked gain in weight appeared, reaching a maximum of 79 kg. The distribution of fat and the type of mammae and abdomen corresponded to a "hypophysial" adiposity. About two years before death, a polyuria set in, the amount of urine having been normal up to that time. Following this, there was a decrease in weight so that after an illness of four and one-half years, the patient weighed 33 kg. and she had a cachectic appearance.

The clinical diagnosis made by Weygandt was of a tumor in the right peduncle. Later, Troegle, on the basis of the changing symptomatology, made the diagnosis of a serous meningitis.

Necropsy revealed a tumor at the base of the brain between the optic tracts and the cerebral peduncles. The infundibulum was not visible, and the hypophysis lay compressed in the sella turcica. The lateral ventricles were dilated, and the corpus callosum and the fornix were thinned out. The tumor was found to be an irregularly shaped cyst which was infiltrating, and whose boundaries could not be clearly limited. Anteriorly, it compressed the pallidum and internal capsule. Posteriorly, it compressed the pallidum in its lateral portion, and since it grew more mesially, it had almost entirely destroyed the right thalamus in its anterior portion, and had strongly compressed the left thalamus. The tumor was no longer visible at the level of the pulvinar. Besides this tumor, the brain showed also two peculiar cavities about the size of a plum, which were connected neither with one another, with the ventricles, nor with the tumor. The first of these began in the right thalamus, laterally, and above the tumor and extended back into the pulvinar. The second began in the substantia nigra; it lay mainly in the right part of the pons and extended backward to the beginning of the fourth ventricle. These cavities seemed to be pushing the brain substance aside rather than destroying it.

Histologic examination of the cysts showed them to have a wall made up of fine glia fibers without astrocytes and with a few gitter cells which were filled with lipoid or with pigment. The vessels in the vicinity were not increased in number. The tumor showed a more complicated picture. In most areas, it consisted of a thick network of a fibrillary ground substance with numerous cells. It looked like a cell-rich fibrosarcoma with the hematoxylin-eosin preparation. The nuclei were long and oval in form and moderately large; they contained a fine network of chromatin, with a lack of a nucleolus in most cases. With the van Gieson preparation, the fibrils were a light yellowish brown, fine and of uniform thickness and ran in parallel bands. The fibrils were arranged in bundles which ran longitudinally, transversely and obliquely. The vascular element in the tumor was significant. The tumor was not solid, however, because here and there one found lacunae of various sizes, and indeed in some places the fibrillar structure of the tumor was completely gone. In other parts of the tumor, "amorphous concrements" were found which stained intensely with hematoxylin. Specific stains for connective tissue structures showed nothing.

Typical neurinomas are usually tumors of the peripheral nervous system, are usually multiple and often in large numbers (von Recklinghausen's neurofibromatosis). "There is no doubt, however, that typical neurinomata occur also in the central nervous system." Centoni, in 1917, reported thirty-seven cases of neurofibromatosis with central changes; in eight of them, he believed there was a central neurinoma. Molter and also Kirch lately reported similar cases. "A solitary neurinoma of the brain is until now unknown in my experience and in this respect our case is unique."

The author then turns to the question of the origin and nature of the cavities which were found at necropsy. According to his view, the walls of these cavities had neither reactive growths nor glia elements, and therefore could not be looked on as simple cysts of softening. They are developmental anomalies due to a fetal disease of the brain which took place before the fibers were formed. In this respect, he quotes experiments of Spatz, who showed that in lesions of the cord in new-born animals, the signs of destruction disappear more quickly and easily than in the adult, and particularly that the products of destruction disappear completely and without evidence of connective or glia elements. There is formed a "porus" with a simply built wall. Spatz called attention to the importance of this finding in explaining porencephaly and syringomyelia. The author feels that the case cited above is one of porobulbia so far as the two cavities are concerned.

Clinically, the author believes that probably long before the severe clinical signs appeared, there was evidence of a circumscribed tumor of the brain. By this evidence the assumption of the early appearance of the porus is sustained. The sudden amelioration of the symptoms on a few occasions during the course of the illness is explained by the fact that the contents of the cyst were either absorbed or suddenly emptied into the ventricles, in this way producing complete relief. The endocrine disturbance was in the nature of a dystrophia adiposogenitalis though not of a typical kind.

ALPERS, Philadelphia.

PSYCHOSES IN INFECTIOUS DISEASES, ESPECIALLY TYPHUS AND RELAPSING FEVER. SKLIAR, Monatschr. f. Psychiat. u. Neurol. **52**:21 (July) 1922.

The author reports the results of his observations on psychoses occurring during the course of certain infectious diseases. The material consisted chiefly of cases of typhus fever and relapsing fever, with scattered cases of typhoid, influenza, malaria, pneumonia, acute articular rheumatism, cholera and hydrophobia. He classifies them as follows:

A. POST-INFECTIOUS PSYCHOSES

1. The ordinary chronic psychoses; the manic-depressive psychoses, praecox and paretic psychoses, occurring incidentally during an infectious disease. The number of such cases was considerable.

2. Amentia cases, which differ in no respect from the general run of amentias.

3. The so-called asthenic delirium — characterized by a clouding of consciousness, various hallucinations and delusions and motor restlessness, which lasts a few days, and is followed by an amnesia for the entire period of the psychosis. These disturbances have all the characteristics of the dreamy state — we refer to it as asthenic merely because it follows an exhausting disease.

4. Manic-delirious states — very transient disturbances, which show, on the one hand, manic symptoms (elevated tone, flight of ideas, logorrhea, motor restlessness, exalted delusions) and, on the other hand, delirious features (dulness and clouding with subsequent amnesia). Neither the asthenic delirium nor the manic-delirious state can be classed as specific psychoses, characteristic of any infectious disease; they were the same in all the diseases. The author has observed similar states following hunger exhaustion.

5. During the convalescent period there occur psychoses with symptoms of organic brain disease: unequal pupils, diminished or absent light reaction, increased knee reflexes, dysarthria, impaired memory, apathy and indifference. At first the symptoms suggest paresis, but many of the patients make gradual recoveries after a few months. A few die of exhaustion; a few run a chronic course, remaining apathetic and indifferent, with diminished movement, slow speech and lack of bladder and bowel control. For convenience we can call these cases postinfectious pseudoparesis. But similar disturbances occur after any exhausting illness or disease, and so cannot be classed as specific infectious psychoses.

B. Infectious Period

1. We have noted psychoses of short duration in the prodromal stages of relapsing fever. They are quite similar to the asthenic deliria of the convalescent period. They are referred to in the literature as "initial deliria," and described particularly in typhoid and smallpox.

2. Similar disturbances, of short duration, were noted in relapsing fever during the crisis, lasting a few hours to a few days, and followed by amnesia. These deliria all have the general character of the dreamy state; as they occur in various infections, they cannot be considered specific for any one infectious disease.

3. Psychoses which develop during the febrile period, usually at the height of the fever, and disappear with the disappearance of the fever. These are the so-called fever deliria. The author feels that they are characteristic of a state (febrile) rather than of any particular infectious disease.

Thus we find various psychoses occurring during infectious diseases. During the infectious period, we may have initial delirium. During the convalescent period, we find the asthenic delirium, the manic delirious state, the amentias and the pseudoparetic and dementia forms. All these types of psychoses can occur as a result of other causes, such as exhaustion, alcohol, etc., and are the same in all infectious diseases. Some diseases, such as typhus fever, have a more severe course and produce more severe pictures than others. But there is no specific infectious psychosis characteristic of each infectious disease.

With regard to the effect of heredity, the author finds little relation between inherited tendency and the occurrence of febrile and postfebrile psychoses.

What is the main factor — fever, infection, or exhaustion? The author recognizes that fever is an important factor (witness the delirium in sunstroke), but believes that it is not the only factor, because in certain diseases, such as influenza, malaria and erysipelas, delirium occurs with moderate temperatures, while in tuberculosis even high temperatures fail to produce delirium. Further, the febrile delirium sometimes persists after the fever has dropped to normal. Further still, the delirium in some infectious diseases is much more severe than in other diseases with similar temperature. All these points suggest that the infection is an important factor as well as the fever.

In the postinfectious psychoses, exhaustion plays an essential rôle—exhaustion brought on by the toxemia and the fever.

Regarding the pathologic basis of the various psychoses described, there is a difference of opinion. Various changes have been found—such as acute edema, punctate hemorrhages, small encephalitic foci, as well as some degenerative changes in cells and fibers. The author feels that as the psychoses in which these changes are found are identical with those of other infectious diseases, with no demonstrable anatomic changes, there can be no essential connection.

SUMMARY

1. Pathologic anatomy cannot explain the psychoses occurring in infectious diseases. Hydrophobia may be an exception to this rule.

2. There is no such thing as a definite infection psychosis in the sense of a specific disease.

3. Each stage of the infectious diseases has definite, but not specific, psychoses: these psychoses are identical with others arising from other (noninfectious) causes.

4. During the febrile stage, there are the "febrile deliria," dependent in part on the fever, in part on the toxemias. In certain diseases, such as typhus, there are certain manifestations of organic change (paretic and Korsakow types) dependent on the special toxin. Perhaps the pathologic changes found in the central nervous system in these cases have something to do with this. There are few diseases in which with low or absent temperatures (malaria, certain stages of smallpox, etc.) the toxins alone are responsible for the psychic manifestations. Similarly, initial deliria occur in certain diseases (typhoid and relapsing fever) while the temperature is still normal. These disturbances, which Kraeplin calls "initial deliria," are transient, violent, epileptiform dream states, in which a sudden access of toxins causes a shock to the central nervous system. During the critical fall of temperature in certain infections (relapsing fever, pneumonia), there are transient dream states brought on by the sudden change (drop) of temperature, and classed as "critical" deliria.

5. During the convalescence, there are transient asthenic deliria, manic-delirious states (which are really deliria with manic features which may last one or two weeks), more protracted forms of amentia, hysteria and other psychogenic disturbances, and protracted postinfectious paretic and dementia states. All these mental disturbances occurring during convalescence are due chiefly to exhaustion of the central nervous system after protracted illness. This is especially true in the pseudoparalytic and dementia states, especially in typhus fever.

6. Inherited taint or psychopathic constitution plays a small rôle—certainly a much smaller rôle than in the psychogenic psychoses following emotional strain (war, imprisonment, etc.).

7. The ordinary psychoses (dementia praecox and manic-depressive insanity) can also occur during infectious diseases.

SELLING, Portland, Ore.

CLINICAL AND ANATOMIC FINDINGS IN A CASE OF CARBON MONOXID POISONING. H. PINEAS, Ztschr. f. d. ges. Neurol. u. Psychiat. **93**:36 (Sept.) 1924.

The author reports the case of a woman, aged 62, who attempted suicide by means of gas. She had been inhaling gas for about fifteen minutes when

she was found by her husband, and, in about ten hours, regained consciousness. Thirty days after the poisoning, she came to the author. When first seen, she showed slight cyanosis of the face and more marked cyanosis of the lips. The left palpebral fissure was smaller than the right. The pupils were equal, reacted well to light and convergence, and the eye motions were full in all directions. The fundi were normal. At rest, the left nasolabial fold was less marked than the right. The tongue was brought only to the lips, and then quickly withdrawn. Passive movement of the arms showed rigor in all the joints, more marked, perhaps, on the left than on the right. Active movements were possible, but with lessened power. Both arm reflexes were active and equal. The abdominal reflex was not obtained. Passive movements of the legs showed no definite rigidity. Active movements were possible. The Achilles, patellar and plantar reflexes were normal. No pathologic reflexes were present. Sensation was intact as far as could be determined. There was bladder and rectal incontinence. The heart was slightly enlarged to the left, the tone was pure, and the action of heart and pulse was irregular.

The patient was akinetic in all her movements. She did not respond to questions, and made continual movements of mastication and smacking of the lips. When the pulse was palpated, she attempted to withdraw her hand; in doing so, she held the arm motionless for a moment, then carried her hand to her mouth and wiped something away from her tongue. On being told to lift both arms above her head, she did not respond, and, when shown how to do it, the patient carried first her left hand and then her right to her forehead, and then began to scratch her head. The right arm then fell back on the bed, and the left remained in a position as if for scratching. On being told to shake hands, she refused first, then gave her right hand, the grip being first slight then stronger. The left hand remained in the meantime on her forehead. On inqury as to whether she understood the questions, she replied in the affirmative. Responses to all commands were similar to the foregoing. When her hands were placed on her head and she was commanded to put them on the bed, she first refused, and then wiped her mouth with her right hand and laid her hand on the bed. On being tested for stereognosis, she took up all the objects and made scrawling movements with them on the table. She could identify no objects.

The patient remained in bed one week, making no spontaneous movements of her head or body. She died about seven weeks after the poisoning.

In his discussion, the author points out that the patient showed a psychomotor disturbance which has been called by Kleist a "psychomotor apraxia." He thinks that he is dealing in this case with an apraxia, that the inability to do things is not due to a lack of understanding because of the patient's assertions to the contrary, and that paresis is out of the question because the patient could carry out active motions. The case shows a symmetrical disturbance in motion, the patient lying quietly on her back for the most part and refusing to speak. The trunk and neck muscles were most involved in the process, for the arms and legs could be moved. Besides the "psychomotor apraxia," the patient exhibited "hypermetamorphic motions" (Wernicke) called in the modern terminology, pseudospontaneous or motions of perseveration. Wernicke describes these as "compulsive primitive motions in which the patients seize, taste, point or tear at all objects which are at hand." Besides these, Kleist observed catalepsy, vasomotor excitability and outbreaks of perspiration. All these the patient exhibited. The reaction which the patient showed in withdrawing her hand when her pulse was felt, the author looks on as a "pseudo-negativism."

Where are the seats of the lesion in a process which produces these phenomena? Kleist first placed the lesion in the striatum, but in a later publication, he thought the hyperkinetic phenomena were due to a lesion of the frontocerebellar pathways and to changes of unknown intensity which extend into the basal ganglia (thalamus, putamen, pallidum). Stauffenberg called attention to the similarity between psychomotor apraxic symptoms and those occurring in disease of the striatum. On the basis of studies on frontal lobe tumors, he gives lesions in the left frontal lobe as causes of psychomotor disturbances. Kleist built up his theory largely on the observation of psychomotor phenomena, whereas Stauffenburg dealt more with akinetic symptoms. The lesions in both cases, however, were in the basal ganglia, so that Kleist's idea, according to the author, that bilateral disease of the basal ganglia is responsible for akinetic psychomotor disturbances is fulfilled. In the author's case, a bilateral softening of the pallidum was seen at necropsy. Both putamina showed numerous small areas of destruction. The vessels in these regions were very much dilated and the walls thickened. All other regions of the brain were intact. On the basis of these findings, the author asserts that all the clinical findings — akinesia, muscle rigidity, amimia, catalepsy, mutism, parakinesia and hyperkinesia with perseveration symptoms and outbreaks of sweating, may be explained by the bilateral softening in the pallidum and the lacuna-like degeneration in the putamina. The first four symptoms — akinesia, muscle rigidity, amimia and catalepsy — correspond to the "akinetic-rigid pallidal syndrome" of the Vogts and Foerster. On the ground of Kleist's theory, the author feels that the hyperkinesia and parakinesia are symptoms of basal ganglion disease. They are caused by bilateral "striatum-plus pallidum injury."

In the second portion of his paper, the author discusses the clinical course of carbon monoxid poisoning. The number of clinical descriptions of the course of carbon monoxid poisoning is relatively small, because most patients die before any signs develop. Lewin, about one hundred years ago, described a case in which the muscles of the left side were contracted, while the right arm moved constantly. Another patient who was unconscious made continuous movements of flexion and extension with his right arm. Still another patient threw his body forward and backward, flexed and extended his fingers continually. Ruge recently studied twelve cases of carbon monoxid poisoning, and in one of ninety-five days' duration, he reported spastic resistance of the arms on passive motion, while the legs were paralyzed. Necropsy showed areas of softening in both lenticular nuclei. Richter, in 1922, reported two cases similar to that of the author. Clinically, these cases showed a high grade of rigidity, accompanied by staring facies, slowness of movement, tendency to maintenance of abnormal positions, lack of extrapyramidal hyperkinesias (choreiform and athetotic motion), and, in one case, speech disturbance of the cortical type (paraphasias and perseveration), and in another case of the subcortical type, dysarthria. These cases resemble in very great degree that of the author. The paper ends with a discussion of the difference between Richter's cases and the author's and with a question as to the relation between "psyche and basal ganglia."

ALPERS, Philadelphia.

RESEARCHES ON THE PHYSIOLOGY OF THE NERVOUS SYSTEM IN THE HUMAN FOETUS. M. BOLAFFIO and G. ARTOM, Arch. d. scienze biol. **5**:457, 1924.

The authors, after a brief account of the work already done on the physiology of the nervous system in the fetus, report the results of their experiments

carried out on fetuses at different periods of gestation. The smallest fetus studied by them was 5.5 cm. long. Their conclusions are as follows:

There exist in the human fetus, at least up to the third month, muscular reflexes under the guise of slow contractions which tend to spread and become general. As the fetus develops, these contractions become more rapid, and their tendency to spread diminishes, until toward the beginning of the sixth month, this tendency disappears. Up to about the eighth month, the muscular reflexes prevail over the tendon reflexes, which in their turn prevail in the mature fetus. They are produced independently of the central nervous system, but are inhibited by a mechanism (inhibitory) which has its seat in the cerebral hemispheres.

The tendon reflexes in the fetuses appear after the fifth month. We could not elicit them before that time. At first they are few and slow. Little by little, as the development advances, they become more apparent, and are present in full during the last two months of the intra-uterine life. In the fetal period there also exists an inhibitory mechanism for the tendon reflexes, which has its seat in the cerebral hemispheres.

The abdominal reflexes can be elicited during the last two months of intra-uterine life; but they are inconstant, almost always asymmetrical and easily exhaustible. The cremasteric reflexes are occasionally present during the last two months, but they are usually few and asymmetrical.

The plantar reflex is obtained between the sixth and seventh month. From that time up to birth, there are present two forms of this reflex, namely: a superficial (cutaneous) plantar reflex, which manifests itself by a slow extension of the big toe and a slow extension and abduction of the other toes; and a deep plantar reflex (muscular or osteoperiostic), which manifests itself by a slow flexion of all the toes. From the seventh month up to birth, we observe the grasping reflex, which is a deep reflex. Inconstant, variable reactions take place following superficial stimuli of the palm.

The corneal reflex can be observed at the seventh month. At birth, it is constant and lively. The conjunctival reflex is almost constant.

The pupillary reflex to light can also be observed at the seventh month. At birth, it is constant but slow.

The sucking reflex appears during the sixth month. Its reflexogenous zone consists then of the mucosa of the gums. As growth progresses, it becomes more active, and its reflexogenous zone spreads and includes also the external surface of the lips. Up to the sixth month of fetal life, the cerebral cortex and the pyramidal tract cannot be excited by mechanical and electrical stimuli, whereas the pons, the bulb and the cord can be excited. At birth, the cortex of the motor area may be excited. The fetal brain exercises an inhibitory influence on the muscular and tendinous reflexes.

The nerves of the full grown fetus are not susceptible to mechanical stimuli, with the exception of the median nerve, which rarely offers a slight reaction. Nothing positive can be said about the mechanical excitability of the nerves of the fetus before the end of gestation.

The nerves and, to a lesser degree, the muscles of the fetus are hypo-excitable to electric currents. Their contractions are variable, but in general they are rapid for the galvanic, prevalent at the cathode, and slow for the faradic. The nerves and muscles of the seventh month fetus react to an electric current in the same manner as those of the full grown fetus.

NACCARATI, New York.

A Clinical Study of Diseases of the Circulation of the Extremities: A Description of a New Method of Examination. Barney Brooks and Fred A. Jostes, Arch. Surg. **9**:485 (Nov.) 1924.

By the use of a carefully calibrated thermocouple, molded into the shaft of a needle, and a galvanometer, the authors succeeded in obtaining accurate temperature readings in the tissues of the extremities. The purpose of their study was to determine the rapidity and amount of temperature changes in a limb following the application and release of a tourniquet. As the freedom of the circulation of the blood controls the rapidity of the temperature rise following the removal of a constriction over the blood vessels leading to the extremity involved, they felt that by careful estimation of the temperature changes following release of a tourniquet, the efficiency of the arterial supply could be determined.

From the results obtained in thirty-seven examinations of eighteen persons, it would seem that the study of the temperature changes in the tissues resulting from alternate obstruction and release of the arterial circulation gives valuable information as to the presence or absence of arterial disease. Obviously, the temperature of the distal tissues of a normal extremity will fall if the circulation of blood is completely obstructed, and will rise if the obstruction is removed, assuming that the extremity is exposed to ordinary room temperature. In all persons examined whose arteries were presumably normal, the temperature of the distal tissues fell during the period of obstruction and rose immediately to approximately its former level, after the removal of the obstruction. In all persons examined whose arteries were known to be obstructed by disease, the temperature of the distal tissues fell during the period of obstructed arterial circulation, but did not begin to rise immediately after the release of the obstruction. In all instances, the fall in temperature in the distal tissues continued for a period of from two to seven minutes after the removal of the obstruction. In one instance of very marked arterial disease, no rise in temperature was observed. The length of time elapsing between the removal of the tourniquet and the beginning of the rise in temperature of the distal tissues seems to be an index of the magnitude of obstruction of the large arteries. Another factor considered is the rapidity of the rise of temperature after it has once begun. In all instances of presumably normal circulation, the rise has been rapid, from 1 to 3 degrees C. per minute. In most instances in which the arteries have been known to be diseased, the rise has been less rapid.

It would seem, therefore, that the rapidity of the rise of temperature might be an index to the condition of the circulation in tissues in a sense independent of the condition of the main arteries of the extremity. In the case of patients receiving repeated examinations, the curves obtained have been similar. Thus, it would seem that the form of the curve obtained might be characteristic of the condition existing. If there is a continued fall of temperature in the distal tissues after the tourniquet is removed, obstruction of the large arteries exists. The significance of the rate of rapidity in the rise in temperature after it has once begun is not now clear. Further study will be necessary to differentiate the significance of these two factors. Grant, Philadelphia.

The Effect of Malaria on the Nervous System with Special Reference to the Malarial Psychoses. Clement B. Masson, Am. Jour. Med. Sc. **168**:334, 1924.

Mental symptoms have been observed in cases of malaria since the earliest history of the disease. The malignant tertian organism is the most frequent

cause of cerebral manifestations of the disease, yet other types of plasmodii also cause mental changes, and there are many other associated factors that are productive of mental symptoms. In studying the literature and his own cases, the author has grouped his mental observations as follows: 1. Initial delirium with or without coma. By many writers, the acute delirium is thought to occur only in the alcoholic person; this, while usually the case, is not always true. The delirium may come before the fever, it may be very light, it may be dreamlike, it may be severe, such as a delirium tremens, an incoherent delirium with professional hallucinations, or an agitated delirium with epileptiform convulsions. The coma which follows may be intermittent with the fever or may pass on to death. 2. Typical psychoses that may accompany the attack or follow it. Some writers hold that no definite psychosis develops without predilection. Pasmanic and Gehrenstein find depression the most common sequel. The author concludes that any type of psychosis may develop according to the type of patient, the heredity and the severity of the infection. Confusional states, amnesia or even complete dementia may follow. The amnesia may be characteristic, dating from the time of the initial attack, and recurring with each secondary attack. This last occurs especially in cases of chronic malaria. 3. The pathology of the nervous system in malaria which cannot be given exactly because of divergent views. Of outstanding importance is the plugging of the small arterioles and capillaries in the brain and spinal cord. This tendency to thrombosis and embolism causes many serious changes. There is a marked hyperemia of the pial vessels, causing congestion of the subarachnoid, perivascular and pericellular spaces, which in turn causes obstruction of the capillaries and leads to the formation of punctiform hemorrhages. There is a "so-called" toxic change in the cells. 4. Cerebral malaria with aphasia. This is said not to be uncommon and is explained by the plugging of the cerebral vessels. 5. Other neurologic manifestations such as neuralgias, either simple or multiple. Paralyses of the vagus center and heat center are reported. Upper and lower motor neuron palsies are found, as well as monoplegias, hemiplegias and cases simulating anterior poliomyelitis. Severe headaches and Korsakow's psychosis also indicate cerebral involvement. Tremors of all kinds are found. 6. Neurasthenia with emotionalism, depressions, irritability and vertigo. This may follow the disease, or the convulsions may be made much more severe and frequent by malaria. 7. Hysteria. This may be provoked by the disease or may be greatly aggravated.

Finally, the author states that in twelve definite psychotic patients at St. Elizabeth's Hospital the acquisition of malaria caused no change in the symptoms; he then reports seven cases studied which show that the malarial psychoses may assume any form, depending on the personality. Delirium, coma, confusional states and amnesia are the most common varieties.

WILSON, Clifton Springs, N. Y.

STHDIES ON THE STRUCTURE AND DEVELOPMENT OF THE MESENCEPHALON. LUIGI CASTALDI, Arch. Ital. d. anat. e embriol. **20**:23, 1923; **21**:172, 1924.

With these two papers, the author has begun the publication of a vast monograph on the mesencephalon. His purpose is, as he states, to reorganize the enormous mass of bibliographic material about this complicated portion of the encephalon, paying particular attention to its anatomo-comparative-embryologic segments, and then to make a complete study of its structure in one single species, *Cavia cobaya*, from its first embryonic stage up to its adult

form. The methods of Cajal, Nissl, Weigert, Marchi, Giemsa and Golgi-Cox were used in his researches, which were conducted with a great wealth of material.

The reviewer finds that it is impossible to assemble all the data cited by the author and those discovered by him, and to give here his minute criticism of the various gray nuclei and bundles of the mesencephalon. No item which bears on the anatomy of the midbrain has not been considered.

In the first paper, the author gives a general description of the gray matter of the mesencephalon. He mentions its numerous nuclei, regrouping some of them and describing others found by him (dorsal nucleus of the posterior commissure, large tegmental cells of the lateral lemniscus, dorsomedial and dorsolateral nuclei of the central gray matter, etc.). In this part, he also discusses the problems of the olfactory and visual pathways of the mesencephalon, making a distinction between olfactory and para-olfactory pathways and giving a new embryologic and experimental contribution to Edinger and Beccari's para-olfactory lobe. He studies the formations of the habenular and mammillary systems which have to do with the mesencephalon, and in this study, as in all the chapters in his work, he considers their histogenesis, their myelinization and their definitive structure. A comprehensive view of the various bundles of fibers which pass through the mesencephalon longitudinally is also given.

The author then proceeds to study the anterior corpora quadrigemina, their layers, the pathways leading to and from them, especially the descending tegmental ones. He determines the time of their formation and their various relations. In this chapter, experimental investigations also bring new contributions to the knowledge of the visual layers and the suprageniculate nucleus. A special chapter is devoted to the transverse peduncular tract.

In the second paper, the author deals at length with the nuclei of the third and fourth pair of nerves, with those of the medial longitudinal fasciculus and those of the posterior commissure. An elaborate discussion serves to cast light on the confusion existing on this important subject. The author does not consider the neurobiotaxic factors as the only forces operating in the phenomena of approach between the nuclei of the third and fourth nerves, and among those of the two sides. He shows that the modifications of form that take place in the encephalon are also to be considered as important factors. Finally, he confutes Bok's theory, which claims that the fourth is a visceromotor nerve.

The two articles occupy about 300 pages and contain ninety-four figures. On the whole, considering the wealth of material used by the author and the great quantity of bibliographic news, they represent a conscientious and authoritative research work, whose completion will be warmly welcomed by the neurologists.

NACCARATI, New York.

THE AMPHIBIAN FOREBRAIN. I. AMBLYSTOMA, EXTERNAL FORM. II. THE OLFACTORY BULB OF AMBLYSTOMA. C. Judson Herrick, J. Comp. Neurol. **37**:361 (Oct.) 1924.

These two papers continue the author's studies of the organization of the amphibian nervous system. The cerebral hemispheres of the salamanders have assumed the general form of those of mammals, but reduced to lowest terms consistent with that form. There is no differentiated cerebral cortex, though the regions where the cortex appears in reptiles can be recognized. What physiological and morphogenetic factors have operated in the fabrication of cortex within these regions?

In previous papers, the sensory pathways leading from the periphery to the correlation centers of the midbrain and thalamus have been analyzed as a preliminary to an understanding of the functional systems of nerve fibers which enter the correlation mechanisms of the cerebral hemispheres. Fibers from the diencephalic centers and fibers from the olfactory bulb come into physiologic relationship within the cerebral hemisphere, and the analysis of these relationships should reveal the physiologic organization within which cortex was differentiated. As introductory to this analysis, the first of these two papers presents illustrations of the external and ventricular sculpturing of the forebrains of *Amblystoma* and *Cryptobranchus*. The second paper describes the internal structure of the olfactory bulb of *Amblystoma*.

The olfactory bulb of *Urodeles* forms a large part (in some cases as much as one third) of the lateral wall of the hemisphere. The outermost layer of the bulb, or layer of nerve fibers (here called the implantation cone of the olfactory nerve), is outside the brain and is separated from the glomerular layer by the external limiting membrane of the brain wall. This membrane is perforated by fascicles of fila olfactoria. The ependymal elements of the bulb span the distance between internal and external limiting membranes of the brain wall, to both of which they are attached. The mitral cells and granule cells of the bulb are of simpler form than in mammals, and their arrangement suggests some reflections on the probable functional relations and phylogenetic history of these elements. The amphibian granules are regarded as structurally unpolarized and capable of transmitting nervous excitations through their two widely branched processes in either direction, thus reinforcing inactive mitral cells through collateral discharges from those which have been activated directly from the periphery.

C. J. Herrick, Chicago.

Reactions to Light in the Earthworm, Lumbricus Terrestris L. Walter N. Hess, J. Morphol. **39**:515 (Dec. 5) 1924.

Lumbricus terrestris is negative to all except weak light, to which it is positive. Light-adapted specimens are positive to stronger light than dark-adapted specimens. Specimens that have been excessively stimulated, by contact or by heat, are positive to stronger light than are specimens that have not been thus stimulated. Stimulation of earthworms posterior to segment five causes them to respond negatively, provided the cerebral ganglion is functional, but no such reactions are obtained by the stimulation of the posterior three fourths of the body. Sexually mature specimens are positive to stronger light than are sexually immature specimens. One pair of nerves from the cerebral ganglion supplies the prostomium, one pair from the middle region of the circumpharyngeal connectives supplies segment one, two pairs from the ventral region of the connectives supply segment two, while segment three is supplied by three pairs of nerves from the subpharyngeal ganglion. Each succeeding segment, except the last, receives three pairs of nerves from its own ventral nerve cord. Removal of the cerebral ganglion causes earthworms to become definitely positive in their reactions to lights of moderate intensities. A similar, though less evident, condition is brought about by the separation of the lobes of the cerebral ganglia or by the removal of one of them. The severing of nerves that supply certain segments has a similar effect on the reactions to light as the removal of the segments that these nerves supply. The results show that the organism is most sensitive to light

in the region of the prostomium and that photic sensitivity decreases gradatim posteriorly from this region for a considerable distance. Specimens in which the ventral nerve cord has been severed are apparently double, so far as their reactions to light are concerned. The portion of the worm in front of the incision is definitely negative, while that behind the incision is positive. The results lead to the conclusion that the cerebral ganglion is the controlling factor in negative reactions.

Areas of greater and lesser sensitivity were plotted, by means of localized light, over the entire body of the earthworm. The results obtained confirm those of the previous experiments and also show that for any particular segment the most sensitive region is in the median dorsolateral areas. A study of the epidermal cells in the photosensitive and the nonphotosensitive areas has revealed the presence of a certain type of cell in the former, but absent from the latter areas, indicating that they are photoreceptor cells.

WYMAN, Boston.

CONTRIBUTION TO THE STUDY OF THE PITUITARY SYNDROMES. L. FERRANNINI, Arch. di patol. e clin. med. **2**:13 (April) 1924.

The author describes a syndrome of infantilism and diabetes insipidus in a boy, 14 years old, who had signs of involvement of both the anterior and posterior lobes of the pituitary gland, which, however, as the roentgen examination of the sella turcica revealed, was not definitely increased in size.

A thorough presentation of the case is given from the morphologic standpoint (Viola's anthropometric method) and from the biochemical standpoint, with particular consideration of the hydric exchange. In relation to the anterior lobe the patient showed: deficient stature, short and thin bones, lower extremities relatively long, arrest of development of the genital organs, and absence of the secondary sexual characteristics, rough skin, and well developed intelligence. In relation to the postpituitary, the most essential sign was hydruria (from 14 to 19 liters of urine in twenty-four hours), in which the hydric excretion through the urine and the feces was superior to the amount of water introduced through the aliments. This was accompanied by polydipsia, which, if not satisfied, gave rise to serious phenomena of abstinence, intense intestinal stasis, marked increase in the elimination of chlorids, with alteration of carbohydrate metabolism. The polyuria was only temporarily reduced by the administration of pituitary extracts, especially by hypodermic injections of postpituitary preparations.

After the elaborate critical review of the pathogenic theories of diabetes insipidus, the author concludes that while in his case there was evidence of a lesion of the neurohypophysis, in many other cases diabetes insipidus depends on a lesion of the centers for diuresis located in the optopeduncular region and in the floor of the third ventricle. These centers function under the stimulus of the hypophysial secretion which reaches the same by way of the infundibulum and the tuber cinereum. The diagnosis of the seat of the lesion in diabetes insipidus must be made in each clinical case from the consideration of the particular individual symptoms and of the concomitant syndromes, because polyuria may occur if the hormonic stimulus is lacking, if it cannot reach the centers of the diuresis because of defective canalization, or if the centers do not react to stimulation because of lesions within them.

NACCARATI, New York.

THE LABYRINTHINE TESTS IN CEREBELLAR DISEASE. L. SELLING and F. B. KISTNER, J. A. M. A. **81**:1194 (Oct. 6) 1923.

In order to clear up certain responses in which the interpretation has not been clear, the results of a careful study in a small group of cases was undertaken by the authors.

An outline of value to be derived from the Bárány test based on the study of four cases is offered. It is to be borne in mind that supratentorial lesions manifest themselves indirectly, whereas lesions in the posterior fossa give rise to direct disturbance of semicircular canal function. The responses derived from each group are as follows:

A. Supratentorial Tumors:

1. Exaggeration of responses from brain stem (nystagmus) or cerebellum (past pointing) or both.
2. Diminished responses from the brain stem (impaired or perverted nystagmus) especially from the vertical canals.
3. Presence of all responses from the cerebellum.
4. Diminution of certain of these cerebellar responses.

B. Cerebellar Tumors:

1. Spontaneous of one arm in one direction with loss of reactive past pointing in the opposite direction or loss of reactive past pointing in one arm in one direction without spontaneous fault.
2. Pressure symptoms in the brain stem, lost, impaired or perverted responses, especially from the vertical canals, less often from the horizontals as well.

C. Cerebellopontile Angle Tumor:

1. Loss of responses (or gross impairment) from the cochlear and vestibular nerve on the affected side.
2. Pressure symptoms of the brain stem — lost, impaired or perverted responses especially from the verticals, less often from the horizontals.
3. Pressure symptoms of the cerebellum (homologous hemisphere) with impairment of past pointing in the homolateral arm, but retention of the reactive past pointing.

FAY, Philadelphia.

GROWTH AND DEVELOPMENT OF THE NEURAL PLATE IN THE ALLANTOIS. VERA DANCHAKOFF and ANNA AGASSIZ, J. Comp. Neurol. **37**:387 (Oct.) 1924.

The embryonic nervous system from its first appearance, if cut out from the hen's egg and transplanted into the chorio-allantois entire or in part, continues to develop. No appreciable growth is observed if a part of the primitive streak or even the whole primitive streak is grafted previous to the organization of the embryonic cells into a neural plate; if isolated at this stage, embryonic cells seem capable only of maintaining themselves. The neural plate consists of cells which, though structurally not differentiated, nevertheless are strictly limited in their potencies. They develop in a predetermined way, irrespective of whether they remain in the normal position or are transplanted. Irrespective of whether the neural plate is transplanted as a

whole or as a part separated from one or both of its ends; of whether it remains in the graft on the surface without closing or is separated from the ectoderm and entirely surrounded by mesodermal elements; of whether it even becomes split by sagittal cleavage into branches — its cells behave as self-determining units. They exhibit a high power of proliferation with differentiation never failing to accompany it. So intimately do these two aspects of embryonic cell activity seem to be associated that neither the one nor the other has ever been observed separately in these grafts. No analogy seems to exist between isolated but normal embryonic cells and tumor cells. Ciliated ependymal cells, neuroglia cells, neuroblasts, typical nerve cells with processes assembled into stems, spinal ganglia, and accumulations of sympathetic ganglionic cells, outgrowth of their processes to a great distance, splitting of the nerve stems into tiny branches—all these structures and processes are exhibited in the grafts independently of any relation to the other parts of the organism, which in these preparations are missing. In contrast with the predetermined differentiation of the cells of the neural plate, the general organization of the cellular material into the typical structure of the normal spinal cord seems to be dependent on various external factors, different for every phase of development.

C. J. Herrick, Chicago.

Mixture of Paranoid and Depressive Symptoms in Psychoses of Advanced Years. Seelert, Monatschr. f. Psychiat. u. Neurol. **52**:140 (Sept.) 1922.

Previous studies by the same author tend to show that the depressive and the paranoid psychoses of advanced years should not be considered as separate entities. The essential pathogenic processes are the same, or similar, in the two. The two types cannot be sharply differentiated. Mixed forms occur, and the basis for these mixed forms lies in the constitutional basis on which they develop.

The author reports in detail a case in point: a senile psychosis showing both paranoid and depressive features, and in which the past history of the patient before onset of the psychosis shows well developed paranoid and depressive trends. He points out the variability in the clinical picture of the senile and presenile psychoses, and maintains that these variations are not a sufficient basis for differences in classification. To evaluate these variations properly, we must recognize the fact that they are dependent on fundamental differences in make-up. From this point of view, he disapproves of such classifications as presenile delusions of persecution, senile depressive insanity, involutional paranoia, involutional paraphrenia, presenile paraphrenia, etc.

On the basis that persons of advanced years with paranoid psychoses do not recover, whereas those with depressive psychoses do, Kehrer concludes that there are different relationships between the exogenic and endogenic factors in the two groups. The author disagrees. According to his view, the endogenic factor is the determinant. Pathologic states depending on an endogenic-depressive or manic-depressive constitution have a tendency to disappear; those which develop on a paranoid constitution rarely do. When we come to the depressive and paranoid psychoses of advanced years, we find this same difference in course. This merely demonstrates that the endogenic factor determines the picture and maintains its general type and tendency, even when associated with organic changes in the brain.

Selling, Portland, Ore.

INFLUENCE OF THYMUS ON BODILY GROWTH DETERMINED BY THE BIOMETRIC METHOD. LUIGI CASTALDI, Atti della Società Italiana per il Progresso delle scienze, XII Meeting, April, 1923.

Following the same biometric methods already employed in his original researches on the influence of the thyroid and the suprarenal cortex on the various phases of growth of the body, the author has made a statistical study of a thousand thymus weights in man. Placing them in series according to the function of the age, he found that the absolute weight of the human thymus, after a rapid and great increase in the prenatal period, gradually diminishes, reaching its farthest point between 1 and 2 years of age, and then it increases again, rising to its highest point between the ages of 11 and 15 years. From that time the involution, which is continuous with the exception of a slight increase between the ages of 61 and 65 years, begins, the persistence of the thymus in the adult being not at all an inconstant variation. The ratio of the thymus weight to the bodily weight is higher in the prenatal period, and attains its highest point at birth either in relation to the bodily weight or in its percentage (thymic index), or by finding the quantity of thymus per kilogram of bodily weight or per centimeter of stature. Treating these statistical data with biometrical methods, the coefficient of correlation of the thymic weight in function of the bodily weight is found to be the highest during the fetal period ($r = 0.837$), then it descends rapidly up to 5 years (0.268), to rise again successively (0.376), and again descends anew to its minimum after puberty. Analogous results were obtained in the calculations in the function of the stature.

All these facts, according to the author, prove that the thymic value in relation to bodily growth is greatest in the prenatal period, and in the postnatal period it is greater during the prepubertal epoch of greatest peripubertal bodily lengthening; that is, the behavior of the thymus is opposite to that which he found in the study of the thyroid. This antagonism between thyroid and thymus with respect to their influence on the somatic growth coincides perfectly with the findings of experimental morphology.

NACCARATI, New York.

NEUROBLAST VERSUS SHEATH CELL IN THE DEVELOPMENT OF PERIPHERAL NERVES. ROSS G. HARRISON, J. Comp. Neurol. **37**:123 (June) 1924.

In this paper, Harrison presents in detail the evidence for conclusions which were announced in more summary form many years earlier. In an extensive series of crucial experiments, it is demonstrated that axons are formed only by outgrowth from the cell bodies of neuroblasts and never by the fusion of sheath cells or other elements. The sheath cells have nothing to do with the formation of the axon, which develops before sheath cells are present, and which may continue to develop when the source of all sheath cells has been experimentally destroyed. The sheath cells are not of mesodermal origin, but arise for the most part from the ganglion crest in connection with the spinal ganglia, though some may arise in later stages by emigration along the ventral roots. The sheath cells are regarded as the neuroglia of the peripheral nerves.

The operations on which these conclusions are based include the removal of the neural crests and dorsal half of the spinal cord of frog larvae shortly after the closure of the neural tube, the removal of the ventral half of the spinal cord, leaving the dorsal half and neural crest in place, and finally

the removal of the entire spinal cord after the neural crest has begun to differentiate. In the last case, sensory nerves with sheath cells are formed in the normal way, but motor fibers are lacking. In the second case also, motor nerves fail to develop, though sensory nerves with sheath cells are present. In the first case, no spinal ganglia, sensory nerves or sheath cells develop, but motor nerves develop normally, except that their fibers are not accompanied by sheath cells. The sheath cells for both the sensory fibers and the motor fibers come exclusively from the neural crests in the earlier stages, and chiefly so at all stages.

C. J. Herrick, Chicago.

The Ketogenic Diet in the Treatment of Epilepsy. M. G. Peterman, Am. J. Dis. Child. **28**:28 (July) 1924.

After Geyelin (Med. Rec. **99**:1037, 1921) described the favorable effects of prolonged fasting in epilepsy, Wilder (Mayo Clinic Bull. **2**:307, 1921) noted that a characteristic metabolic irregularity accompanying fasting is the accumulation and excretion of aceto-acetic acid and its products, beta-hydroxybutyric acid and acetone; and further that these substances are also formed in excess in diets in which carbohydrate and protein are sufficiently restricted. He suggested, accordingly, that a metabolic result similar to fasting could be obtained by a restricted carbohydrate and protein (ketogenic) diet. Any favorable result on epileptic seizures with this diet or during starvation appears to be in large part traceable to the accumulation of aceto-acetic acid which is fat-soluble and anesthetic.

Peterman plans his ketogenic diet as follows: The metabolic requirements of the patient are determined; 1 gm. of protein for children and two-thirds gram for adults are allowed per kilogram of body weight; 10 to 15 grams of carbohydrate per kilogram of body weight are given, and enough fat is added to make up the required calories.

Of seventeen patients treated with a ketogenic diet, the observations covering periods of two months to one year, ten are entirely free from convulsions, four are greatly improved, two patients could not be followed, and one failed to respond. The most favorable results were obtained in cases of petit mal, which form of epilepsy, the author notes, does not respond well to phenobarbital. The author suggests the need of further studies, and advocates a strict daily regimen in all cases and phenobarbital and sodium bromid when indicated.

Vonderahe, Cincinnati.

Clinical, Anatomic and Pathogenic Notes on Juvenile Diabetes Insipidus. M. Pincherle and L. Magni, Arch. di patol. e clin. med. **3**:261 (Sept.) 1924.

This is a study of five cases of diabetes insipidus in young persons, two of whom died from acute intercurrent disease, and came to necropsy. The authors, after a short but complete exposition of the different theories (endocrine, nervous, mixed) of diabetes insipidus and of the five clinical histories, give the anatomopathologic findings in the two cases, which seem to confirm the duality of the causes of the disease advanced in some of the pathogenic theories.

In one case they found serious and extensive lesions of the hypophysis, with signs of active inflammatory reactions and advanced sclerosis, together with moderate neuroglia proliferation and slight degenerative lesions of the tuber cinereum. In the other case in which diabetes insipidus had been

caused by what turned out to be a mild form of epidemic encephalitis, extensive and very severe degenerative lesions of the ganglionic cells of the tuber cinereum were found accompanied by intense phenomena of neuronophagia and perivascular infiltration, while the pituitary gland did not show any histologic alteration. These two diverse findings have led the authors to conclude in favor of the dual neurohypophysial conception in the pathogenic interpretation of the disturbed hydric exchange which is the basis of diabetes insipidus. In the majority of such cases the hypophysis or the tuberal centers or the important connections existing between the gland and the latter are altered, either separately or at the same time.

NACCARATI, New York.

EYE STRUCTURE IN NORMAL AND EYE-MUTANT DROSOPHILAS. O. A. JOHANNSEN, J. Morphol. **39**:337 (Dec. 5) 1924.

The ommatidium of the normal eye of *Drosophila melanogaster* and *Drosophila virilis* differs from those of *Musca* and *Calliphora* in that the secondary pigment cells lie approximately in a single plane, the nucleus of the displaced retinula lies relatively more distad, and the rhabdomeres are relatively farther apart. Two pigments, wine-red and ochre-yellow, are present. The eyes of the eye color mutants, eosin-miniature, eosin-vermilion, pink, purple, ruby, sepia, tinged, vermilion, and white, have the same structures and pigments as those possessed by the normal eye, differing only in the amount and distribution of the pigments. The pigments are much reduced in tinged, eosin-vermilion and in the female of eosin-miniature, and they are absent in white and in the male of eosin-miniature. Most of the elements of the normal eye are also present in the mutants glazed, spineless-glass, and wax, although in the first the rhabdomeres are more strongly developed than in the normal, while in the others they are either not distinctly differentiated or absent. In all of them, the corneal facets are more or less deformed. The pseudocone, shortened in glaze, is small in spineless-glass and entirely wanting in wax. In most respects glaze shows least modification. The eye structure in all three forms suggests an irregular growth of the cells of the pupal eye without a corresponding perfecting of the imaginal characters.

WYMAN, Boston.

THE CORTICAL LAMINATION PATTERN OF THE OPPOSSUM, DIDELPHYS VIRGINIANA. PERCIVAL ALLEN GRAY, JR., J. Comp. Neurol. **37**:221 (Aug.) 1924

The histologic study of this cortex by Brodmann's method reveals upward of twenty fields that can be anatomically distinguished by the patterns of cellular lamination. Most of these areas can be recognized also in Weigert preparations. The limits of these areas are charted on surface views of the brain, and the cytoarchitecture of most of them is illustrated in section. The hippocampal cortex and pyriform cortex are elaborately developed; the neopallium shows the mammalian structure, but greatly simplified, and in many of the areas very atypical as compared with that of man. The excitable area of this cortex had previously been explored physiologically in cooperation with Turner, and the histologic structure of this excitable cortex shows some interesting peculiarities. The motor cortex is commonly described as lacking the granular layer (IV of Brodmann). Most of the excitable field of the opossum possesses a well developed granular layer. This applies especially to those centers found to be associated with the control of skeletal muscles

which lie mostly behind the orbital (so-called cruciate) fissure. In front of this fissure is an agranular cortex which was found to be associated only with the control of muscles of the face and vibrissae.

C. J. Herrick, Chicago.

The Occurrence of Herpes Zoster in Hodgkin's Disease. Henry K. Pancost and Eugene P. Pendergrass, Am. J. M. Sc. **168**:326, 1924.

The occurrence of herpetiform eruptions in Hodgkin's disease is rare. The authors found only one case reported in the literature. Several cases of malignancy, however, complicated by herpes zoster are reported. The theories of causation of herpes are then reviewed. It has been shown that irritation from a tumor may cause herpes, but no cancerous infiltration of the nerve itself has been found. Head and Campbell have demonstrated the profound inflammatory changes in the posterior root ganglion. They also state that secondary deposits of malignant disease attacking the spine may produce a rash indistinguishable from that of herpes zoster. The authors, then, report four cases of Hodgkin's disease and one case of ovarian sarcoma complicated by herpes. Necropsy was not performed in any of the cases, but biopsies were made in four of them, and the findings in each case are conclusive. Judging from these cases, the authors conclude that the severity of the disease had nothing to do with the appearance of the rash, but that the occurrence of herpes zoster is frequent enough to be classed among the skin manifestations of Hodgkin's disease.

Wilson, Clifton Springs, N. Y.

An Experimental Study of the Cerebral Physiology of the Virginian Opossum. Fred T. Rogers, J. Comp. Neurol. **37**:265 (Aug.) 1924.

In harmony with other recent students of the question, Rogers finds that electrical stimulation of the cortex evokes localized movements of the facial, shoulder, arm and neck muscles, but none for the abdominal, hind leg or tail muscles. Extirpation of the excitable cortex without injury of the corpus striatum is followed by transient difficulty in using the contralateral hand and by more enduring weakness. Destruction of this cortex and the underlying striatum leads to profound paralysis of the opposite forearm and hand and contracture of the neck. Electric stimulation of the corpus striatum fourteen days after destruction of the excitable cortex produces elevation of the head and extension of both forelegs. Unilateral destruction of the occipital cortex with minimal damage to the thalamus is followed by blindness of the contralateral eye and no detectable disturbance in the ipsolateral eye. There is no hemianopsia. Report is made of other operations, including complete decerebration in front of the thalamus, transection between superior and inferior colliculi and lesions of the cerebellum.

C. J. Herrick, Chicago.

Cholesteatoma of the Third Ventricle with Bilateral Argyll Robertson Pupils. F. R. Ford, J. A. M. A. **82**:1046 (March 29) 1924.

An interesting case is reported whose symptoms suggested a suprasellar growth. At operation, a well defined cyst containing cholesterin crystals was disclosed, and later a necropsy showed a tumor which occupied the region of the third ventricle.

The condition is usually associated with tumors of Räthke's pouch and is epithelial in character.

No field defects were noted, although the disks showed 4 diopters of swelling. The patient showed the usual signs of intracranial pressure as well as a paresthesia and paresis of the right foot.

The pupils did not react to light, although vision was present, and the author refers to several similar cases in the literature. The symptom is unusual, but may have decided value in lesions such as this.

FAY, Philadelphia.

THE NUCLEUS OLFACTORIUS ANTERIOR OF THE OPOSSUM. C. JUDSON HERRICK, J. Comp. Neurol. **37**:317 (Aug.) 1924.

The anterior olfactory nucleus is defined as a portion of the secondary olfactory area which retains its primitive undifferentiated character, in contrast with the more specialized secondary olfactory nuclei whose differentiating features are in large measure correlated with their nonolfactory connections. This region of the opossum is described in detail. It extends far forward, adjoining the posterior face of the bulbar formation. When followed backward, it quickly assumes cortical structure on both the lateral and medial sides of the olfactory crus. These cortical sheets, the pyriform and the hippocampal, are in actual contact at their rostral ends with the bulbar formation, and they both receive axons of mitral cells. This cortex is, accordingly, separated from the peripheral olfactory surface by only one synaptic junction, that of the glomeruli. The final section of the paper includes some reflections on the archipallium and the morphogenetic problems suggested by its architecture.

C. J. HERRICK, Chicago.

Society Transaction

SOCIÉTÉ DE NEUROLOGIE DE PARIS

Fifth International Annual Meeting, at the Salpêtrière, Paris,

May 30 and 31, 1924

Multiple Sclerosis

Clinical Aspects.—Professor Otto Veraguth (Zurich): Recent investigations in Switzerland, although unfinished, indicate that there is probably one case of multiple sclerosis to every 3,000 inhabitants, an incidence greater than that of neurosyphilis. The classical triad of symptoms described by Charcot—nystagmus, scanning speech, and intention tremor—occurs only in about 10 per cent. of the cases. Multiplicity of symptoms is the rule, and differential diagnosis may be difficult. Fever may be present in the acute attacks. Manganese poisoning may closely resemble multiple sclerosis, but a history of exposure to manganese can be obtained in these cases. Hysteria may imitate this disease, but Babinski's sign will usually differentiate the functional from the organic disease, although the extensor plantar reflex may be temporarily absent in multiple sclerosis; nothing is easier for the hysterical person to imitate than the Babinski phenomenon. Other conditions to be considered in the differential diagnosis are: epilepsy, paralysis agitans, hemiplegia, lacunar degeneration of Marie, tuberous sclerosis, extracortical axial aplasia, Wilson's syndrome, cerebral tumor, cerebellar atrophy, hereditary ataxia and Friedreich's disease. Spinal types of multiple sclerosis may be confused with syringomyelia, cord tumor, subacute combined degeneration and myelitis. In fact, the old term, "spastic spinal paralysis," certainly indicated at least three common conditions: spinal syphilis, cord tumor and multiple sclerosis.

Except in the classical form of Charcot, multiple sclerosis may simulate the most diverse diseases and may lead to many errors of diagnosis. But certain criteria of this disease are relatively certain: (1) the multiple distribution of lesions indicated by the symptoms, (2) the evolution of the disease by exacerbations interrupted by remissions, (3) the absence of abdominal reflexes, (4) dysmetria and (5) optic atrophy. These five appear in the majority of cases, but not by any means in all.

In the spinal fluid, Oberholzer of Zurich is observing the spectrographic changes according to the method of Professor Victor Henri. These seem to show, in the region of the ultraviolet rays, traces of inorganic substances in solution, perhaps due to myelin degeneration. Possibly this method will give a diagnostic criterion.

There are two explanations of multiple sclerosis: (1) that it is a primary, essential sclerosis (the "periaxial sclerotic encephalomyelitis" of Marburg); (2) that the sclerosis is secondary to an inflammatory process (Erb, Marie, Borst). To me the inflammatory process appears the more plausible. For one thing, primary degenerative diseases of the nervous system are rare, and multiple sclerosis is common; also primary degenerations are familial or hereditary. We certainly do not yet know anything about the means of transmission—Steiner's theory of transmission by a tick is supported by little evidence—and epidemiologic studies, though suggestive, give no real clue.

The bacteriologic work is indefinite, and it seems to me that in all the positive results reported there is little that can withstand the criticisms of such competent bacteriologists as Noguchi and Dörr.

It is often difficult to explain the symptoms of multiple sclerosis on a physiologic basis, for the lesions found at necropsy may not give an anatomic explanation of the disturbed function. If, however, one thinks of the different degrees of disturbance or excitability which may follow a lesion, either in the affected region itself or in nearby intact areas, von Monakow's theory of "diaschisis" satisfactorily explains the great variability of the clinical manifestations. In this way, we can explain disturbances in neurons far from the lesion but functionally connected with the injured neurons.

Treatment is at present tentative. Injections of calomel, thiosinamin and arsphenamin have been tried with varying success. The researches of Stern of Geneva show that preparations like arsphenamin probably cannot pass the choroid plexus. As a rule, patients suffering from multiple sclerosis do not withstand surgical operations well. Rhizotomy is perhaps an exception, and may give good results.

PROF. GEORGES GUILLAIN (Paris): Next to syphilis, multiple sclerosis is the most common disease of the nervous system. The patient is usually from 20 to 30 years of age, and the primary attack passes without much attention, the history eliciting merely a story of slight and transitory difficulties in walking, paresthesia or vertigo. Some months later, the patient has fatigability, difficulty in walking, unsteadiness, stiffness of the legs, disagreeable paresthesias of the hands and feet and perhaps difficulty in starting micturition. To the foregoing symptoms may often be added vertigo, diplopia and diminution of visual acuity. When the patient is examined, no muscular weakness is found, but a slight dysmetria often disturbs the delicate movements of the limbs. The tendon reflexes are exaggerated, there is clonus, a bilateral Babinski reflex and abolition of the abdominal and palatal reflexes. Objectively, there are no sensory disturbances, except a diminution of the vibratory sense in the feet. There may be nystagmoid jerkings and hippus. The spinal fluid shows a negative Wassermann reaction and a positive colloidal reaction. Such is the common picture. After two or three weeks of rest, the symptoms may disappear, only to return in one, two or three years. Then slowly, with each exacerbation, the spastic paralysis increases.

Symptoms related to the vestibular nuclei are common, vertigo, nystagmus, tinnitus, nausea and forced movements being found. Deafness and lesions of the cochlear apparatus have not been observed. No typical psychic state is associated with multiple sclerosis. The sympathetic system and the endocrine glands are not affected. The "classical form" is seen in about 12 per cent. of the cases. This consists of unsteady gait, intention tremor, scanning speech, exaggerated tendon reflexes, abolished abdominal and palatal reflexes, nystagmus and optic atrophy.

The form with "ocular onset" is important, with its progressive diminution of vision or perhaps sudden amaurosis due to a retrobulbar neuritis. This alone, with exaggerated reflexes and a positive cerebrospinal fluid reaction to colloidal tests, may make the diagnosis. Sphenoidal sinusitis must be ruled out. The "pure paraplegic form" is common and may be confused with syphilis, but here again examination of the cerebrospinal fluid will make the differential diagnosis. Lymphocytosis is rare, and an increased albumin content is not constant; the globulin reaction is usually negative, and the Wassermann reaction is always negative. The colloidal gold and colloidal benzoin reactions are of interest; the latter is positive in 63 per cent. of the cases; a

typical benzoin reaction made with the fifteen tube technic would read "001000120001100."

Prof. Jean Piltz (Cracow): I have frequently observed paresthesia in the legs; this was usually most marked in the toes, diminishing in the feet and legs. Loss of position sense may be found in the same distribution; vibratory and tactile anesthesia are not rare.

Professor Long (Geneva) emphasized the possibility of confusion with tumors of the cord when making a diagnosis.

Dr. André Thomas pointed out the importance of a history of remissions in making the diagnosis; the disease is intermittent. The symptoms may vary greatly from time to time, and suggestibility may play a rôle.

Professor Barré (Strasbourg): Symptoms referable to the cerebellum and the vestibular nuclei are common, vertigo, titubation, head movements and nystagmus being frequent. The pure paraplegic form is less common than is usually supposed; a careful history will elicit cerebellar symptoms, transient periods of incoordination being indicated by a history of inability to manipulate small objects. Pain, when present, is central in origin. The cerebrospinal fluid pressure may be raised.

Dr. Lagrange (Bordeaux) discussed pupillary reactions.

Professor Velter gave an historical review of the disease.

Professor Claude told of his psychiatric experiences, saying that multiple sclerosis was never a cause of psychosis.

Dr. Souques emphasized the insidious onset, and discussed the spinal fluid chemistry.

Dr. Babinski said that an extensor plantar reflex was a practically certain indication of organic disease, and that an imitation of this reflex by an hysterical patient could always be recognized and differentiated from the true reflex.

Pathologic Aspects.—Prof. Georges Guillain: The areas of sclerosis are the end result of a diffuse degenerative process, and should not be studied as the central point in the pathology, for their locations cannot be narrowly correlated with the clinical symptoms. The three places of election of these plaques are perivascular, subpial and subependymal. As pointed out by Bertrand, these are the dumping grounds of the products of degeneration of the central nervous system. In the old plaques of two or three years' duration, there is fibrous neuroglia. This does not touch the axis cylinders, which are diminished in number, reduced in size and demyelinated. No products of active degeneration are found. In the recent plaques, all the processes of degeneration may be seen. There are much lipoid débris and many phagocytes. Bertrand has clearly demonstrated that the process of degeneration takes place in each neuronal unit separately; beside a demyelinated axon may be a perfectly normal fiber. Outside the plaques, one may find a pathologic condition, especially in the contiguous areas. These areas are more important than the plaques, which really are scars. The axons are attacked early, and are swollen, the edema separating the neurofibrils; or they may merely lose their staining affinities. The myelin sheaths are altered and surrounded by phagocytes. The vessels show perivascular infiltration with round cells and phagocytes, a result of eliminating the products of degeneration. Secondary degeneration of the Wallerian type may be found, usually showing as a pallor of the corticospinal tracts. A terminal type of diffuse demyelinization has also been observed.

The clinical characters of the disease and its pathology both indicate a diffuse infection of the nervous system. This infectious origin was first maintained by Marie in 1887. I am of the opinion that multiple sclerosis is due to a specific organism, as specific as that of rabies, epidemic encephalitis or syphilis. But it has no relationship with syphilis.

Dr. Bertrand showed lantern slides illustrating his pathologic studies (described above by Guillain). He emphasized the degeneration of the axons and the subependymal and subpial distribution of the plaques.

Dr. Schroeder (Copenhagen) described intranuclear inclusions of rodlike bodies, and illustrated them with excellent lantern slides. He said that he believed the disease to be infectious, probably spirochetal.

Dr. Jumentié showed lantern slides illustrating the pathology, especially the perivascular infiltration, the increased vascularity and meningitis. These points indicated how closely the disease may resemble syphilis. In fact, it is difficult to distinguish it from syphilis both pathologically and clinically.

Proffesor Lhermitte gave an illustrated exposition of the pathology. Meningitis is an important finding, and may even be so marked as to resemble general paralysis. The plaques are often perivascular, and there are also characteristic vascular changes. The reaction about the spinal and cranial nerve roots is also interesting, especially the optic and acoustic nerves. In the cord, lesions are often found at the junction of the spinal nerves and dura. This observation is in line with that of Nageotte on syphilis, and suggests that the virus is in the spinal fluid. Considering the vascular changes, the general pathology and the lesions in other organs, it would seem that the disease is a special infection.

Professor Long (Geneva) presented case histories and showed pathologic specimens to illustrate the difficulty of correlating the two.

Dr. Marquezy told of his experimental work with blood and spinal fluid from nine acute cases injected into twenty-one rabbits and one monkey. The results were negative, both for lesions resembling multiple sclerosis and for spirochetes.

Dr. Targowla discussed the "benzoin colloidal" reaction, and showed typical results. The reaction is read in sixteen tubes; sample reactions in multiple sclerosis are: 0010022221000000, 0220221000000000 and 1221221000000000. The Wassermann reaction is always negative, and the cell count and albumin content are usually normal.

Professor Mestrezat discussed the importance of chemical study of the spinal fluid to indicate the nature of the process of degeneration and the absence or presence of organisms.

Dr. André Thomas reviewed the pathologic findings and pointed out the periaxial degeneration, the tumefactions of the axons and their disappearance. He said that at times there is perhaps an abortive regeneration. The degenerations were illustrated with lantern slides of longitudinal sections. He questioned whether the benzoin reaction proved the absence of syphilis, and whether multiple sclerosis had been scientifically proved to be a disease distinct from syphilis.

Professor Léri discussed familial cases, and considered them as evidence of contagion rather than of inheritance.

Dr. Meige closed the meeting with the announcement that the annual reunion next year will be in June, the subjects for discussion being amyotrophic

lateral sclerosis and migraine. Both of these diseases were of particular interest to Charcot, and this meeting will celebrate both the centenary of Charcot's birth and the twenty-fifth anniversary of the founding of the Société de Neurologie de Paris.

NEW YORK NEUROLOGICAL SOCIETY

Stated Meeting, Dec. 2, 1924

E. G. Zabriskie, M.D., *Presiding*

A Case of Undifferentiated Myoclonus. James H. Huddleson.

A Jew, 23 years old, a stock clerk, complained of constant twitching and jerking of the whole body, with muscular fatigue and difficulty in mental concentration. Except for a neurotic mother, his heredity was negative for three generations. His personal history was negative, except that he reached only the fifth grade in school on account of truancy; his behavior was not antisocial, and his make-up not abnormal.

The present illness began gradually in April, 1920, two weeks after a fall of 5 or 6 feet, which caused a slight scalp wound. Since their onset, the twitchings had become progressively more pronounced. He now had generalized clonic spasms, about sixty to the minute, resembling those of Sydenham's chorea, but more rapid; they appeared anywhere, at irregular intervals, and generally involved a muscle group or several adjacent or symmetrical groups. Among the most frequent were contractions of the abdominal walls, clenching of the fist with flexion at the wrist and elbow, and grimacing of no definite pattern. The spasms immediately increased in any part of the musculature brought under examination; still they never disappeared entirely. The lower extremities were least affected. No fibrillary twitching was observed. Myotatic irritability was diminished.

The deep reflexes were markedly diminished, especially in the arms, in which there was hypotonia; this was not present in the legs. The epigastric and abdominal reflexes were slightly diminished. There were no paralyses and no atrophies. The pupils were normal at rest and in reaction, and although extra-ocular muscles occasionally twitched, there was no nystagmus. There were no ataxias. Deep and superficial touch and temperature senses were normal. The special senses were unimpaired. A mild left hemihypalgesia was demonstrable.

There was no psychoneurosis beyond the symptoms already noted, and nothing psychotic. The mental level was in the borderline group. Mild underdevelopment and undernourishment were seen; the general physical status was otherwise negative. The blood, spinal fluid and urine were normal.

This case is now believed to be one of generalized degenerative tic, although paramyoclonus multiplex remains a possibility. It has resisted all forms of psychotherapy, chemotherapy and physiotherapy.

DISCUSSION

Dr. G. A. Lawrence: Have any metabolism studies been made?

Dr. L. P. Clark: I should like to ask Dr. Huddleson whether he has given any particular medication in this case. If large doses of chloral are given to such patients, and they can stand from 25 to 40 (1.6 to 2.6 gm.) grains, by

gradually increasing the dose one may be able to eliminate the most disturbing myoclonic movements and thus be able to make any kind of physical examination that is desired. In my several studies on the association disease, myoclonus epilepsy, I so modified the myoclonic movements as gradually to eliminate the different kinds of muscular movements, and thus was able to determine which muscular systems were most involved by those movements being last repressed. Lundborg and Hunt have shown that probably the whole myoclonic mechanism is in greater part due to degenerations of the midbrain. However, there are a number of neuromuscular mechanisms that are probably not specifically degenerative in character to be discovered in parts of the central nervous system other than those of midbrain lesions. The lesions for these other atypical myoclonic syndromes are probably due to degenerative conditions in the cortex. Though one accept the midbrain lesion for much of the myoclonic syndrome, it is still greatly modifiable, as one finds in other conditions, by psychic states. For instance, they are greatly affected by heat, cold, excitement, depression and various emotional factors which when thrown in on this neuromuscular mechanism might distort the whole pattern, though the central midbrain degenerative lesion be organically continued. I should like to ask Dr. Huddleson how long the myoclonus has existed in this case?

DR. HUDDLESON: Four and a half years.

DR. CLARK: Have there been any seasonal variations?

DR. HUDDLESON: Not that I know of.

DR. CLARK: Independent of special medication, many sufferers from the myoclonic syndrome may pass months without myoclonic movements. One of my patients was relieved for eight or nine months at a time without medication, and at another time we had the greatest difficulty to decrease the movements by the continual building up of sedatives; yet with proper living he was quite free from his myoclonus, although he had suffered from marked locomotive affect; he would fall like a log whenever large masses of proximal muscles were involved. I should like to ask whether this patient had good and bad days, in which the piling up of muscle movements became so intense that he had to stay in bed? In two of my cases studied by Dr. Prout, we came to the conclusion that the myoclonus was definitely a cortical disease. Later, our individual findings have been disproved by other contributions to this subject.

DR. I. ABRAHAMSON: As Dr. Clark has emphasized, in all these cases the diagnosis rests between paramyoclonus and a generalized tic. The so-called functional element is conspicuous in this case, but these mutations are common to all forms of myoclonus. I am reminded of a case at the Montefiore Home, a sailor who had a marked form of paramyoclonus multiplex; he had spasms of the platysma, the muscles of the neck and upper extremities. This man, whose muscles were constantly in motion, shaved himself with an old fashioned razor, and he made all sorts of things, such as knotted cord work; he would wait for a pause in the movements and pass the razor across his face, then wait for another pause; he kept this up until he had finished shaving, without the loss of a single drop of blood. It was a remarkable sight to behold; one would almost think that there is a large amount of malingering present. There is some tendency to symmetry in the movements; that is, first one arm and then the other will perform the movement; all forms of cutaneous irritations increase the number and amplitude of the movements. Observation also exaggerates them, but they also go on just the same when the patient is not watched. Some of these have been described as electrical. I think this case

is no doubt one of real paramyoclonus multiplex and not a generalized tic nor any other form of functional disease.

Dr. Joshua Rosett: Recently, I have been interested in myoclonus as one of the effects of voluntary hyperpnea. A number of patients who had exhibited this phenomenon during voluntary deep breathing were subsequently operated on and were found to have extramedullary disease, that is, disease within the vertebral canal impinging on the spinal cord. Other patients in whom myoclonus did not appear during the test of voluntary hyperpnea were operated on, and in these no disease was found on the outside of the cord. The myoclonus which appears in certain patients during deep breathing is, however, of a different character from the myoclonus exhibited by Dr. Huddleson's patient. In the former, a number of muscles are convulsed in such a manner that the contraction and relaxation of each muscle is entirely out of time with respect to similar contractions and relaxations of its synergists and antagonists. The result is that no movement takes place at the joints. In Dr. Huddleson's case of myoclonus multiplex, entire groups of synergists contract simultaneously with the relaxation of antagonists. The result is that the patient jerks the head or the limbs.

I cannot see the reason for attributing the increased twitching of this patient when he was brought into this room to a superimposed hysteria. Concentration of attention is always accompanied by a fixed posture. The latter is accomplished by immobilizing the joints by means of an equal contraction of antagonistic muscles. The act of concentration of attention is therefore accompanied by an increased release of muscle tonus. This patient's attention became concentrated on the audience when he was brought into this room, and the normal increased release of muscle tonus was utilized in him for the production of increased movements. That such was really the case may be easily ascertained by concentrating the attention of a person affected with muscular rigidity, on the one hand, and of one affected with an abnormal movement, on the other. The first patient will then become more rigid, while movements of the second will become increased.

Dr. S. Rothenberg (by invitation): A woman, suffering from tic movements, simulating those shown in this patient, came to me about five years ago for analysis and relaxation treatments. Neither form of treatment did her any good. Finally, she took up Christian science, and that too seemed not to have helped her in controlling these violent movements of her head and body. On four different occasions, these movements caused subluxations of her shoulder joints, which required anesthesia for replacement. The diagnosis of myoclonus multiplex was made by competent neurologists of both Philadelphia and New York. Despite this severe and persistent affliction, she was determined to control her tic movements so that, with another girl, she was enabled to join a theatrical team. They soon were accepted on the main circuit known as the Orpheum circuit, where I myself saw them perform. During the entire time of their singing and dancing, my patient showed no sign of her tic. Though she is unable to control her condition at any other time, she has never had any twitching while on the stage. One certainly must make the diagnosis of myoclonus multiplex with many reservations.

Dr. S. E. Jelliffe: I think Dr. Clark has said everything that was in my mind, but Dr. Rosett started another thought, and so I have been asking myself, watching these movements, what are they trying to do? Dr. Rosett has partly suggested what they are trying to do; it may be that they are trying to overcome some localized alkalosis. Are these movements from that point

of view to be considered as efficient? The muscular masses in human beings phyletically considered are extremely complicated. It is known that polyphasic wavelike neuronic stimuli are constantly playing in functional patterns, which are immersed, so to speak, in the grosser muscular structures. It is not inconceivable that these muscular contractions serve to show that localized functional activity is impaired, and it may be through alkalosis of functional units.

DR. WALTER M. KRAUS: I have had occasion to see one patient with what at first seemed to be paramyoclonus multiplex. He was sent to me at the base hospital with this diagnosis. Electrical examination showed no responses typical of the disease. I finally discovered that if I told him not to move his leg, he would cease doing so. After having examined him three times, his twitching stopped, and I sent him on with a diagnosis of neurosis. The case was obviously purely functional. Last year in Paris, I saw a case showing motility disorder which was diagnosed Unverricht's myoclonus epilepsy. The patient did not show continuous myoclonic movement. He could be made to have an attack by any form of moderately active sensory stimulus. He would then have severe myoclonic seizures lasting for a minute or so. I believe that the case which Dr. Huddleson has reported belongs in the group with Unverricht's myoclonus epilepsy. I noticed the patient's movements in the hall, and they were by no means so active or so extensive as they are now. It is apparent that the emotional excitement of being presented has exaggerated the movements.

DR. HUDDLESON: In regard to medication, this patient has been given some chloral and bromid, but not in the large doses Dr. Clark suggested. He has also taken hyoscine and other sedatives, but nothing that did him a great deal of good. He had received a great deal of physiotherapy and hydrotherapy. He gained a little temporary benefit, but nothing seemed to give him any permanent relief. The question of his having had encephalitis has come up. There is no history of encephalitis, and he had been under observation four weeks previous to the onset. He had been in the army two or three weeks before he had this fall, and then another week or two before the symptoms began. We have not a good history dating back a year or so. So far as we know, the man has not had seasonal remissions. As the patient has been observed, there is little fibrillary twitching but more massive contraction, not only of one muscle but of a group of muscles, and there is general movement of the limbs; I think that is one of the reasons why a diagnosis of paramyoclonus multiplex was not made by most observers. Of course, what Dr. Abrahamson said about the symmetrical movements not being simultaneous in time is correct; the movements are very much the same, but one will begin before the other, and that is true in this case. The patient does not show any nystagmus. Just as I have not known of seasonal remissions, I do not know of any remissions under treatment. As Dr. Kraus said, the movements were very much less while he was standing in the hall than when he was under the observation of several people. They are much worse under observation and vary under all sorts of conditions; but the point I wanted to make was that they never entirely cease, and he has something of the kind even during sleep.

OLD AGE FACTOR IN PSYCHANALYTIC THERAPY. SMITH ELY JELLIFFE.

Dr. Jelliffe prepared this paper, he explained, more for general practitioners than for specialists. The subject was wide. Primarily, what is old age? Some persons are born old; many an old man is still youthful. The chronologic

age is not an index of the physiologic age, and still further, physiologic senescence is not an absolute criterion for psychologic regression. Thus the literati tell us that Conrad began his career at 50, and tradition still tongues the anecdote that Gladstone started to learn Greek when some ten years older. Whom the "gods love" may still "die young," and Mozart, Keats, Shelley and other names spring to the fore as illustrative of the early burning out of some men of genius; yet, on the other hand, many a chronologic dotard has offered to the world surpassing spiritual gifts of enduring social value.

The most orthodox old saw runs that "man is as old as his arteries," and the name of Osler has been widely traduced as saying that "sixty" is the proper chronological stadium for chloroforming. Taken all in all, whereas these generalized formulations have statistics on their side, on a larger review one cannot refrain from the counter attack, apropos of mathematical absolutes, that the three advancing categories, "Lies, damn lies, and statistics" more correctly state the actual truth when the individual himself comes for review.

Advancing age brings with it its inevitable decline. This is the law of life that advances to death, from which no living organism, from ameba to homo, can escape. Bios, the old Greek word, meant both life and death: in which we see repeated throughout the evolutionary scheme of things attraction and repulsion in physics, positive and negative in chemistry, pain and pleasure in sensation, sickness and health in medicine, good and bad in ethics, and comedy and tragedy to the universal onlooker, call "It" by any name one will: "God," "Fate," or "Das Es."

Psychology in its largest vision offers some interpretative formula as to how human beings behave in view of this inevitableness, and this is the theme into which the present paper enters in but a fragmentary way.

Case 1.—A boiler maker, aged 62, competent up to two years before an accident, had regressed to the level of a night watchman on the river front in the employ of a large corporation. One night, he stumbled and fell on an incoming vessel and sustained a trifling injury, somatically speaking. Shortly thereafter, he was discharged, since evidence was incontrovertable that he was not up to his job. Psychologically, however, his reaction was that he had been given a "dirty deal," and since the natural cupidity of his surrounding mediums, wife, sons, etc., supported him, a medicolegal proceeding was inaugurated to show that the injury sustained—trifling abrasions of the body—was the cause of his incompetency. He himself reacted by an excessive exploitation of the injustice dealt to him in his discharge. He dissolved in tears, emotionally overwhelmed by the cruelty of the corporation, and had for months sat in his chair at home, a typical picture of an "arteriosclerotic dement with compensatory paranoid ideas." As a matter of fact, the injury sustained was not a competent producing cause (medicolegal requirement phrasing) of the clinical picture presented, yet this is one of the types of situations in which the "expert," presumably myself, is called on to decide and adjust the relative merits of the situation.

Case 2.—A young woman made a complaint to a policeman in the Grand Central Station that a man, aged 70, sitting by her side, with outspread encompassing newspaper as a shield, nudged her; as she turned in response, she saw his penis revealed, erect or semideclining. The old man was arrested, and his case called for an "expert opinion."

Here the problem falls within a different category. "Pathological Exhibitionism" is the nosologic group into which the case falls. It is immaterial here to give the statistics of this type of abnormal behavior, which must be

dealt with by the district attorney in New York or any other large city. We are, for the moment, only interested in the psychanalytic interpretation of this type of behavior.

In the study of the present instance, we have an old offender, and, sociologically speaking, Bellevue and the Manhattan State Hospital, more or less adequately deals with him; but for our present discussion, the obvious outstanding delict is but an exaggerated example of a vast medley of similar situations varying from the palace to the hovel, to which the "innocent female" is exposed.

How does the psychanalyst deal with these situations, first as an agent of society, and secondly as an interpretor of the instinctive impulses of the individual? In the rôle of an agent of society, he must condemn the sick person, not to Sing Sing, but to the Manhattan State Hospital; but secondly, he is scientifically (sociologically conserving) curious as to the specific causes of the individual's behavior. From this aspect, he may find many ameliorating circumstances, so far as he may discover — and indeed he did unearth in this case — compulsive factors dating from early childhood, which forced him to the conclusion that this type of conduct was not wittingly vicious, but unconsciously compelling. This enabled him to say the patient's behavior was psychotic and sick and not "criminal," as per the "legal definition."

In the earlier years of the development of psychanalysis, the neuroses and psychoneuroses were thought to be the special fields for the applicability of the generalizations with which Freud had made us familiar, but as time went on it developed that a general philosophy of life and its interpretations lay behind the principles so conscientiously worked out by Freud. The "unconscious," long time ago felt as a force underlying instinctive behavior, now came under the scalpel of specific dissection, and it soon became evident that the human being really was worthy of the Nobel prize for his superior gift of "passing the buck" to himself.

Psychopathology gives the general term "rationalization" to this process, and it distinguishes a great variety of grades in it. Some of it is undoubtedly physiologic — for certainly if one did not have a swift cocksure answer to things, but had to inquire into the underlying "isness of the is" — he could not function. Much of the stuff fed us from the cradle to the grave as education, knowledge and logic is a crazy patchwork of these fictions; but as it constitutes the particular gilded small coin of the masses, it passes muster and is serviceable in the turmoil of life temporally. When pathologic rationalizations commence to influence behavior in a manner inimical to the patient's own comfort or that of his surroundings, then the problem arises as to how to get at these inner factors and relieve them.

Freud's and Abraham's contributions are quoted, but the literature is scanty.

Case 3.—The patient, aged 72, had a partial motor aphasia and slight hemiplegia, which had evidently been occasioned by a cerebral softening. The usual advice, etc., was given, and a few months after the acute situation had passed, his physician sent him to me to be looked over. He still had a residual difficulty in finding his words; his hemiplegia had cleared up except for slight weakness. But he was anxious to know how to live; he had had a slightly similar attack a few years previously, and he partly shared the superstition of "three out all out." And so I talked with him from the psychanalytic point of view. It soon was learned that during the past four or five years he had been getting increasingly violently angry, was more and more irritable, and often these explosions left him all in, and the "chronic

chip on the shoulder" that he carried everywhere with him received no relief from golf, motoring, business or anything. He was still an active, forceful personality, and we had to overhaul the whole works — which we did. He was securely well-to-do, hence the "self preservation" aspects of his instinctive life were not deeply involved — but it soon developed that he was handling in an imperfect manner a declining ability to have intercource from nonerection, although harassed by augmenting eroticism. This is a frequent situation in older men, and when made widowers in the sixties, the various clinical pictures resulting are distressing for the patient and baffling for the physician.

In the present instance, we had increasing irritability, increased blood pressure, violent outbursts, psychical, and a couple of arterial blow-outs.

That which was playing on the eroticism was an old daughter complex component. Her relations with the father were cordial and pleasant, but she was not as wise as she might have been in periodically permitting him to get a glimpse of a difficult marital situation with herself. Every time this was brought up, he was extremely kind and helpful — gave her advice, comfort and even money; but it soon became evident that he repressed a hatred to the "son-in-law" and "wanted" the daughter, and the response was an attack of temper, displaced over to the wife, and an increase in the eroticism which by reason of the outburst on the wife found no way of satisfying itself — hence an increasing sense of frustration.

This is but a short thumbnail outline of a complex situation to which the old man caught on and soon established a better rapport with the wife, and regained satisfactory relations; he had it out with the son-in-law and obtained a good adjustment there; this helped the son-in-law over a hard bump, about which he had not wanted to tell his wife. This eased up her friction somewhat, and a much better working combination was established. He is now 80, and busy still. He has had no further blow-outs.

Case 4.—This case related to an old phobic condition in a woman 55 years old when first seen. She had had a psychoneurosis all her life, and for seven years past had been mildly psychotic. Conversion symptoms of swelling, heat and turgidity of the vagina were distressing, and a persistent belief that her nose was growing larger and was the center of all eyes had been very much in evidence. The analysis cleared up the conversion symptoms within a few months, and after two years of treatment carried on irregularly, she made an excellent recovery, now of about eight years' permanency.

Case 5.—A brakeman on the Portland Oregon Railroad came into my office. He was 56 years of age. In direct, simple language he told me that for about ten years he had had a recurrent attack of intense depression every month, which lasted for three or four days. During this time he attended to his work with the greatest difficulty, invariably thought of suicide and had a host of guilty feelings. These guilty feelings chiefly centered themselves on three situations: his relations with prostitute or degraded women, adolescent and manhood masturbation and boyhood bestiality. Careful anamnesis revealed that the periods of depression began approximately a month after his mother's death. He was single; she had been a widow for a number of years, and he had supported and lived with her some years after the father's death, due to a railroad accident. Still further anamnesis revealed that the attacks came on every twenty-eight days and lasted three or four days only, and careful inquiry and a few fragments of unconscious material — dreams — showed that he was reliving, as a substitute for his mother attachment, her menstrual epochs, which lasted three or four days, and in her had been

characterized by periods of intense depression. This mother identification, the Oedipus situation, the time period, and the unconscious situation being carefully gone into, the patient left me. Two years later, I received a letter from him saying that he thought he understood the whole business. He had not had another period of depression and was perfectly well. Whether it is all true or not, I am not in a position to say. I present it as a cameo for the inspection of anyone interested.

Dr. Jelliffe then went on to discuss the failures, which were as numerous as the successes, but the final conviction of an optimistic attitude, since even the failures in therapy showed their reasonableness, and that the neurotic or psychotic adjustment reached by the patient was for him a better compromise than could be offered by "reality." Reality was too hard. In O'Neill's latest expression, "God is Hard."

DISCUSSION

DR. L. P. CLARK: Mr. Chairman, as usual I am delighted with Dr. Jelliffe's lucid and interesting presentation of a psychanalytic subject. I am particularly interested in his sane and clear demarcation of the flexibility of the application of psychanalysis to everyday problems whatever they may be, whether in youth or in age, and especially when it reaches up into the fixed patterns of behavior in the aged. My own experience, at first under the tutelage of Freud, led me to think that we must be chary of handling cases of advanced years, but my general experience has been comparable to that of Dr. Jelliffe's, that we must not pay attention to the chronologic age but to the adaptability of the psychic organ in all its relationships to environment, and if we keep our minds open to the data presented to us, we can accomplish a great deal. As Dr. Jelliffe says, pessimism certainly closes the door to any therapeutic approach. An effort has been made in English speaking countries to reduce psychanalysis to principles of scientific mechanisms, to follow up biologic trends and bring them into accord with structural and static patterns. I have been guilty, with others, in attempting to determine where we could link up various forms of psychoneurotic syndromes with biologic concepts, thus striving for a certain degree of relationship between physiologic behavior and psychic and emotional adaptations. I am persuaded that we have two sharp divisions of psychanalytic effort: The one is to swing back toward a scientific neuromechanism in which certain phases of the dreams are the basic concepts to work on, and the other is to swing clear of psychanalysis as a science and drive into the conception of it as an art, which will take precedence over natural science and is related to the impulse—or instinctive psychology. In the growing use of psychanalysis in the living conditions with which we may have to deal, it is getting to be more and more definitely the first line of attack. It will therefore lose some of the scientific formulations of biologic science but will be nearer to the living art to which psychanalysis must devote increasing attention. I am more and more persuaded that the crux of psychanalysis will be away from a purely mechanical and biologic concept, and more toward an art which is closely related to living social reactions. To some it may be a questionable procedure in methodology to discuss psychanalysis as an art versus its science. One hears it urged that until the practice is better established, it is better to let it rest on the so-called firmer foundation of mechanical principles. Can psychanalysis ever, any more than any of the arts, gain a direct aid from science? Are the two mutually colligated or supportive? We find that all the arts in some indirect and mysterious way are aided by scientific formulations — but not

directly. Assuredly, psychanalysis as an art is not dissimilar in its general rules of growth to those underlying other arts. A science which is based on objective and provable data often stands in the way of the subject-mood and its operation. The cry of the artist is heard over and over again, that science is built on an entirely different stratum of facts; that it invariably stunts the progress of art wherever the former deals with purely logical processes and the latter is basically subjective and intuitive. No one can doubt that psychanalysis purely as a science will be less and less requisitioned in the future consideration of living problems.

(*a*) The Confirmed Stammerer and His Defective Adaptations in the Nursing Period. (*b*) An Interpretation of the Mona Lisa Smile. L. Pierce Clark.

(*a*) Nothing less than a union of biologic, hereditary and individualistic factors can make clear the puzzling etiology of the psychoneuroses of which confirmed stammering must be considered an integral part. No amount of rationalization of the provocative causes gives us any clue to the multitudinous types of speech cramps which are encountered almost at the outset of the confirmed stammerer's career. Within a few weeks, a deficiency in almost all the functions of the oral organs is present. A behavior mechanism of the whole individual organism is involved, and the speech process trips over a previously laid down behavior pattern seemingly arranged for other purposes. What early behavior engrosses the infant which combines the oral organs and the pharyngeal and respiratory functions? None other than the simplest and earliest functions of nursing. One has but to observe any stammerer and the unfortunate use to which he puts these organs in speaking in order to detect all the blurred functions of the act of nursing in operation. I have analyzed several confirmed stammerers, and in two cases I was able to verify from actual repressed data the intensive fixation on the act of nursing and the successive alterations in the process of adaptation to adult speech. Imperfect recovery of infantile memories renders the prognosis poor, as only a complete abreaction paves the way to an emotional healing. Even though the infantile fixation on the breast is fully laid bare, the release of the speech defect is necessarily slow as the perversion of the speech mechanism is deeply ingrained into the most fundamental functions of the whole organism.

(*b*) The general explanation of the manner in which da Vinci conceived the smile of Mona Lisa which he has portrayed in several of his paintings is fairly well stated by Pater. He says that when Leonardo succeeded in reproducing in the face of Mona Lisa the double sense comprised in this smile, namely, the promise of unlimited tenderness and sinister threat, he remained true even in this to the content of his earliest reminiscence. The memory of Leonardo referred to is given by Freud as follows: "It occurs to my mind as a very early memory, when I was still in the cradle, a vulture came down to me; he opened my mouth with his tail and struck me a few times with his tail against my lips." That a person could retain a memory of the nursing period is not impossible, but Freud believes this to be in reality a phantasy which Leonardo formed later and transferred into his childhood. This has remained a fairly reasonable explanation up to the present, but in two cases under analysis it was possible for me to bring into the conscious mind of the patient the actual reproduction of the nursing experience and a recollection of the mother's facial expression during the act.

In the first place, Leonardo was the natural child of Ser Piero da Vinci and was brought up by him. Up to a certain age, we find a general flexibility of emotion in the artist's career, but later, after visiting his own mother, definite impressions seem to have been kindled within him as a result of this visit. It stirred in him an artistic attitude that continually showed in his painting the particular type of facial expression which we find in the Mona Lisa. According to Freud, Leonardo's homosexual constitution was conditioned by an intense infantile fixation on his mother, which in turn was fostered by the passionate devotion that a woman deserted by her lover would naturally lavish on a son. I am suggesting, further, that the famous Leonardesque smile is a subconcious reproduction of the look of complete erotic satisfaction which a woman experiences while nursing her child, and which Leonardo was able to portray through his unconscious memories of his own infancy.

DISCUSSION

DR. KIMBALL YOUNG: While I have made little study of stammering, a case came recently to my notice which brought interesting side-lights on Dr. Clark's thesis. A student in a university nearby had suffered from stammering from the time he was 4 or 5 years old. I discovered, in the first place, that the movements of his mouth when he stumbled over syllables had very much of the nursing movements, and it struck me that it would be worth while to have moving pictures[8] of this type of movement. He had a series of conflicts of a serious compulsive type. He had experienced a certain recurrent dream; in this dream, which came as he said any time he had a conflict of any sort, he was forced to swallow something brown about as large as the end of the index finger. This object would become larger and larger, as big as a room, then as big as a house, as big as the universe, until it became a terrible task to chew it and swallow it. I think this dream is clearly related to the peculiarity of the man's stammering. It is no speech defect in an organic sense. It is not a question of the organic location; it is purely functional. In this case, I was struck first with the likeness to nursing movements and these peculiar dreams which seemed to be associated with his mouth and might be interpretive of the nursing experience. We should have an extended study of the conflicts and compulsions of the functional stammerer. Analysis and such objective records as motion pictures would be worth while.

DR. B. ROSENBLUTH: I have made quite an investigation of stammerers, and especially stammering in very young children, from 3 to 4 years of age. Usually the stammering was associated, practically in all the cases tabulated, with masturbation. The child would masturbate and fall over and relax. Then the mother would come to see what was the matter, and the child would stammer. That happened several times a day, until in time the mother would be unable to trace the masturbation. When these children were corrected from the standpoint of masturbation, they were cured, and others that were allowed to go on longer kept on stammering. This, of course, was hardly noticed until the child began to go to school. There the constant watching of the teacher as compared to the mother made the child continually stammer as a state of defense. Stammerers never stammer when they are alone; only one out of several hundred stammers when alone. I have not looked on it from the angle of the nursing period or that particular hypothesis. As to erotic pleasure which the mother experiences in nursing, I have one case, that of a girl of 18, who took all the babies in the tenement house to her dry breast and derived sexual pleasure.

DR. M. C. ROSE: I have made a study of stammering for some years. Such exercises should be practiced as "Round the rugged rock a ragged rascal ran," and when in the presence of a stranger stammerers will soon get over the habit of stammering. So many people gasp for breath two or three times when they start to make a speech, and have to start in again. If they are brought out and taught good strong games to develop their strength like Roosevelt, and learn a little public speaking, the stammering defect can be overcome.

DR. JELLIFFE: Dr. Clark read a paper a few years ago on stammering, and I repeat some of the things I said then. I firmly believe that Dr. Clark is not quite careful enough in his definition, because I think he will recognize that there are stammerers at different levels, not all are at the breast level by any manner of means; and, as stated, there are stammerers whose speech defects are determined by other genital organizations than the pregenital organization involved in the oral erotic phase. Thus there are easily cured stammerers and others who resist all efforts. Undoubtedly, there are many variations in the patients, but I think that one thing is certain, that Dr. Clark is not quite correct when he speaks of the oral-erotic phase as incorporating other displaced patterns. Some of you may remember that I thought it was quite valid to view the m-m-m-m- type of stammerer as showing oral erotic fixations, whereas the ss-s-s-s- type was evidently showing a displacement from urinary displacement factors; whereas the b-b-b-b- stammerer, especially those who burst out, show a displacement from the analerotic pregenital organization fixation. These displaced patterns as entering into the nosology and etiology of the stammerer are entitled to a little more emphasis than Dr. Clark has placed on them. The scientific investigation of these stammerers is a laborious task; next to the deaf, I think they are the hardest persons to analyze.

The question of "Round the rugged rock" suggested by another speaker is valid, but it is a more or less conscious formula. Whereas the sense of guilt — for there is a sense of guilt when the patients stand on one foot and slink behind — we know perfectly well can be run back along the Oedipus formula in one shape or another. I feel that not enough attention has been given to the respiratory pregenital erotic pattern. Stärcke has recognized this and has dealt with it as the "apnoeic" phase, which possibly antedates in some respects the oral-erotic pattern. The child wants to holler and at the same time to swallow, and it is a 50-50 proposition. Everybody knows that even in advanced years we may hold our breath when we want a good defecation, and everybody knows that this b-b-b-b- has to be suppressed when it comes to the divine afflatus that goes down instead of up.

One word about Leonardo da Vinci. I think Dr. Clark is quite right in some respects. I know at least twenty to thirty clean-cut histories in which the nursing erotic experience is very vivid, and I have known women who have told me that they experienced orgasms which were far more vivid than those derived from sexual relation with the husband.

DR. CLARK: I have taken occasion at least three times to make communications on stammering, and after seven years devoted to the subject I quite agree with Dr. Jelliffe that stammerers are extraordinarily difficult to handle. I have studied both conscious and unconscious data, and my thesis deals essentially with the confirmed stammerer, and not the other types which develop in later life and are allied to collateral diseases. I think essential stammering, like the other phases of ego-erotic neuroses, will be difficult to analyze even if we know the mechanisms by which it evolves, because it belongs to the homosexual constitution, and therefore it is going to be extremely difficult to apply analytic procedure.

CHICAGO NEUROLOGICAL SOCIETY

Regular Meeting, Dec. 18, 1924

HERMAN M. ADLER, M.D., *President, in the Chair*

THE MECHANISM OF THE EMOTIONS. MEYER SOLOMON.

The work of Sherrington, Goltz, Sternberg and Latzko, and others shows that in the absence of the forebrain and even of the midbrain certain emotional expressions may occur without emotional feeling. This is evidence of the necessity of the cerebrum for the existence of emotional feeling, without which there is no real emotion, and for the possible dissociation of emotional expression from emotional feeling. The subjective state is fundamental.

There are two main theories as to the starting point of the emotional state: (1) central — the emotional feeling occurs first, and is accompanied or followed by the bodily reverberation; (2) peripheral — bodily expressions produce the emotional feeling (James, Lange and Sergi theories). The evidence points to the truth of the central theory. This evidence includes psychologic, experimental-physiologic and clinical-neurologic observations. Psychologically, we can detect the presence of feeling at the very beginning, especially in the subtler emotions. In psychopathology, we observe bodily derangements occurring from preceding mental conflicts. Mere mimicry does not directly induce the appropriate mental state.

The evidence from experimental physiology includes the following: (1) Sherrington's dogs not only expressed but actually felt emotion, although only the shoulders, face and head, diaphragm and vocal portion of the muscular mechanism of emotional expression remained connected with consciousness. Therefore, the reverberation from the trunk, limbs and viscera counts for relatively little. (2) Maranon, by injections of epinephrin in certain persons, produced visceral and other bodily reactions much the same as those in spontaneous emotions. In most cases these were but coldly perceived, without the arousal of the subjective state. Therefore, the vegetative phenomena present in emotion are not directly responsible for the initiation of the characteristic mental state.

Under the head of neurologic evidence, Wilson has collected cases of pathologic laughing and crying, even of pronounced degree, which were not accompanied by the appropriate mental state. This shows that emotional expression, skeletal and vegetative, may exist without the appropriate feeling state.

The work of Sherrington and Maranon, and Wilson's cases indicate that the peripheral reactions do not augment the mental state. Evidence points to the conclusion that the mechanism of voluntary activation and of emotional expression is the same, the vegetative phenomena occurring not independently but secondarily to or concurrently with the skeletal reaction.

DISCUSSION

DR. D. M. OLKON: Even if many peripheral stimuli were completely obliterated, it would still be difficult to understand from experiments that emotion is only central and not peripheral as well. For instance, if a dog were blinded and rendered anosmic, it is a question whether there would be any reaction to food in the form of (salivary or gastric) secretion, unless there is another special peripheral sensory mechanism. Experiments along this line

were made at the University of Chicago on white rats. It was found that it took a certain number of trials for a rat to go through a labyrinth in the shortest time. It was also found that when the eyes were removed the rat found its way through the maze to the milk in about the same length of time. The rat still found its way through the maze even after being made deaf. The same result was observed even when the vibrissae were removed and when the paws were anesthetized.

These experiments suggest the possibility of the existence of a kinesthetic sense, a "pull," "tropism" or "force," and that the muscles have certain responsive reactions that enable the animal to orient itself and direct its way through the maze. We are not in a position, therefore, to say that there are no other organs, aside from the "central," which can participate in the integrated reaction. If parts of the periphery are shut off from stimuli and these parts do not react, it still does not prove that all peripheral stimuli have been eliminated and that none of the peripheral organs can take part in the emotional reaction. The experiments cited show that the periphery gave the impetus to start the activity. Emotion cannot be looked on as behavior from a single stimulus of one part of the body mechanism. Emotion, it is said, is merely an instinct seen from the inside and the real cause is to be found in the instinctive response and is always at the perceptual level—the cortex as well as the periphery must join to make an emotion kinetic.

The theory that we are emotional "because we cry" (the "James-Lange" theory) means that it is a selective as well as a mass mechanism. Before a man can be angry, he must be capable of perception and apprehension. Such a concept is not contrary to experimental data. We cannot dissociate and say that either the central mechanism alone or the peripheral alone is the cause of emotion. All that present knowledge warrants us to assume is that both agencies, peripheral and central, together give us the integrated massive response that we call emotion.

DR. JOHN FAVILL: Like most people, I have often noticed following certain stimuli what I call an emotion of fear or anxiety. For instance, being near a stroke of lightning in the woods, a witness to some accident or being suddenly awakened by the telephone at night will give me this emotional response with definite sensations and tachycardia. When I was 24 years of age, I found that I could voluntarily accelerate the action of my heart. I think mine was the fourteenth case in the literature when I reported it in *Heart* in 1917. When I tried to describe it I noted that it gave me sensations identical with those I have mentioned as accompanying fear. With voluntary acceleration, however, there was no idea of danger and no emotion of fear. In other words, I can have the bodily reaction in circumstances that are accompanied by an emotion of fear, and I can have the same bodily reaction, so far as tachycardia and sensations go, in circumstances in which I feel no fear.

DR. GEORGE W. HALL: Dr. Solomon referred to Kinnier Wilson's paper on pathologic laughing and crying. Wilson states that in cases of pseudobulbar paralysis of arteriosclerotic origin the patients weep rather than laugh, whereas in a similar disturbance due to multiple sclerosis they are much more inclined to laugh. There is no doubt that in some organic diseases the patients do not express their real feelings in the emotional outbursts; they may cry when they want to laugh and vice versa. The same is true in mental instability following concussion of the brain. I have records of such patients who state that they may burst into tears or laughter when such behavior does not express their real emotions.

DR. A. B. YUDELSON: Some years ago, a decerebrate dove which had hatched young some weeks before the operation was presented before this Society. After the operation, the dove abandoned her young without evidence of feeling. When the bird was placed on the table and efforts were made to chase it, no expression of fear was manifested. Vision was good, because the bird picked grain from the hand, and hearing was evidently good, but there was no expression of fright.

In postencephalitic conditions, there is great variation in the expression of emotion. A young man with a postencephalitic parkinsonian syndrome, with rigidity of the muscles, especially of the facial muscles of expression, could not betray emotion although he felt emotion. His chief worry was that he had become impotent since having had the "influenza," yet no one could detect any manifestation of emotion or depression on his face. On the other hand, a man, aged 29, with a postencephalitic parkinsonian syndrome, was exceedingly emotional and was able to express his emotion. He was engaged to be married and the young woman decided not to marry him. This led to marked expression of emotion. Another man, aged 33, with a parkinsonian syndrome, came to Northwestern University Neurological Clinic in the hope that something could be done for him by sympathectomy. Dr. Loyal E. Davis did not examine the man, but expressed the opinion that nothing could be done for him. The patient's face was rigid and masklike, and yet he was agitated over his hope. When operation was refused, he broke down and wept.

DR. HERMANN M. ADLER: Dr. Solomon's description of the patients who described their emotions using the words "as if," is of especial interest. I am reminded of the use of this same phraseology by patients in describing hallucinations. The question of differentiating true hallucinations from pseudo-hallucinations may sometimes be solved by consideration of the type of reaction that Dr. Solomon has described. I wonder whether it is proper to compare emotions, from the point of view which Dr. Solomon presents, with hallucinations, the decisive quality of which is a feeling or experience that carries conviction with it.

DR. SOLOMON: In true emotion there is reaction at all levels and hence I divided them into three — the subjective, the somatic and the vegetative; although in true emotion we have response at all levels, in the subtler states we do not have the marked reactions. These subtler states are helpful, by means of introspection and self-study, in showing us that in the early stage we can detect the presence of the subjective feeling state. The idea that I wish to bring home is that in emotion we have an action of the organism as a whole, but from the cortex down; how far down and how marked the bodily reactions will be, depends on the particular emotion and its degree.

Dr. Favill mentioned the voluntary control or acceleration of the heart beat. That is part of the problem of the mechanism of the emotions. I do not believe that any one has direct voluntary control of the heart beat, which is a vegetative manifestation. In cases of so-called voluntary acceleration of the heart beat, there is, preceding it or concomitant with it, a skeletal reaction. There occurs first the assumption of a certain mental and bodily state, and concurrently with this a rapid heart beat; but by mere will we cannot directly make the heart beat more rapidly any more than we can bring about the feeling of heat or cold.

Dr. Hall referred to the quick emotional changes that occur in some arteriosclerotic cases, but there we have a different situation. When we tell the patient sad news, the reaction, even though more rapid and unstable, is

the usual emotional response to this stimulus, and occurs in a normal way. In pathologic laughing or crying, on the other hand, the individual has both the skeletal and the bodily phenomena of true emotional expression but not the appropriate feeling.

Dr. Yudelson referred to the work of Dr. Rogers; his findings do not help in this problem, except that they confirm the conclusion of Sherrington and others that the feeling state is not present if the cerebrum is removed; yet, the other reactions may occur. At the last meeting of the American Medical Association, Dr. Goodhart showed motion pictures of certain post-encephalitic states with apparently purposeless, involuntary movements, which, when slowed up, seemed clearly to have some definite object, design or purpose. I asked him whether he had examined the patients as to the accompanying mental state, and he said that he had not. To me they showed no evidence of emotional feeling, and this would be further evidence that the skeletal and visceral portion may be dissociated from the subjective mental state.

With Dr. Adler, I think the questions of hallucinations and emotional feeling are comparable.

PATHOLOGIC CHANGES IN SPINAL CONTUSION. G. B. HASSIN.

This paper was published in full in the March issue of the ARCHIVES.

DISCUSSION

DR. PETER BASSOE: I admire Dr. Hassin's distinctions between these lesions, which are due partly to direct contusion, partly to concussion and partly to infection. I do not quite understand how he draws a sharp line between contusion and concussion in this case in which there seems to be a difference of degree rather than of kind of lesion. Will Dr. Hassin tell us whether he has read descriptions in the French literature of penetrating gunshot or shell wounds of the spine which cause paralysis without injury to the dura? The bullet had passed outside the spinal canal, and yet there was concussion of the cord. If the histologic details corresponded to those described by Dr. Hassin, I should like to know his opinion.

DR. HASSIN: This presentation was abbreviated, but Dr. Bassoe's questions are answered in the full report. The meningitis was localized, that is to say, was purulent only at certain levels, and, of course, might be responsible for some spinal cord changes. However, meningitis is usually confined to the meninges and spares the parenchyma, which in this case showed widespread lesions. These, as pointed out by Dr. Bassoe, are similar to those described in concussion. Disseminated in both contusion and concussion of the spinal cord, in contusion they are associated with a focal lesion, destruction of a spinal cord segment which can be demonstrated macroscopically. This is not the case in spinal concussion, in which the lesions are, as a rule, microscopic and not focal. The contusion is, therefore, more dangerous than concussion. The presence of the latter may explain such clinical phenomena as loss of the tendon reflexes below the point of lesion. This problem as well as references to literature have been discussed in a previous contribution.

A CASE OF TUMOR OPPOSITE THE LOWER MEDULLA AND UPPER CERVICAL CORD. JOHN FAVILL, D. E. FAXON and F. A. PALMER (by invitation).

A married woman, aged 29, entered Cook County Hospital, August 17. She had been well until April 1, when she had a severe cold. On April 25,

she was frightened by a drunken man and at once developed motor weakness of the left side, more in the arm. There was progressive generalized weakness until July 15, when she went to bed helpless. She had a few days of headache and occasional generalized pain. There was no visual disturbance, no vomiting and no other cranial nerve disturbance.

Examination showed the patient lying in extension and unable to move below the neck. There was spasticity of both arms and legs, increased deep reflexes, bilateral clonus and Babinski sign, with atrophy of the small muscles of both hands. There were indefinite patches of disturbed sensation. The cranial nerves were practically normal throughout. The mental condition was good. There was no fever. Distension of the bladder was marked.

Spinal fluid examination revealed no abnormality and a negative Wassermann test. The blood Wassermann reaction was negative; the urine was normal and a roentgenogram of the cervical spine showed no abnormality. The arm gradually became more flaccid, paresthesia increased; the patient failed rapidly and died on August 25.

The clinical diagnosis was an incomplete transverse myelitis of the cervical region. Necropsy showed an intradural extramedullary tumor springing from the meninges opposite the lower end of the medulla on the right side anteriorly, pressing backward onto the right half of the cerebellum and downward through the foramen magnum. Histologically, the condition was an endothelioma.

DISCUSSION

DR. G. B. HASSIN: I should like to find some explanation for the muscular atrophy which in this case much resembled that seen in amyotrophic lateral sclerosis. The specimen shows that the tumor originated from the dura at the level of the lowermost portion of the medulla or the uppermost portion of the spinal cord. Dr. Palmer, who performed the necropsy, found it, as I understand, within the cranial cavity, just above the foramen magnum.

DR. CHARLES F. READ: I saw this patient before death and know that the condition was as described, and my conceptions of neurologic diagnosis received a decided shock when the postmortem findings were reported. I, also, should like some explanation for the atrophy and the paralytic findings.

DR. JOHN FAVILL: My explanation is that some process interfered with the normal state of the roots. It evidently was outside the substance of the cord, and some transmission of pressure or torsion might explain it; that is the only thing I can think of. The patient denied the presence of pain much of the time.

DR. PETER BASSOE: It seems to me the gross specimen itself offers an explanation. It is unfortunate that, in removal, the cord happened to be torn off at the most critical point. The tumor, as I understand it, was found entirely within the cranial cavity, and yet we have the tumor attached to what is plainly spinal cord and not medulla. It seems to me that the tumor grew just above the foramen magnum and exerted upward pressure so that some of the cord was pulled up into the cranial cavity and became intracranial. The specimen shows at least three segments of spinal cord, and if part of the cervical cord is pulled up into the cranium it is easy enough to understand that the circulatory changes and perhaps a certain amount of cystic accumulation in the arachnoid might explain the atrophy. Another explanation would be that the pulling upward of the cord made traction on the roots and caused the atrophy. The whole thing is mechanical, and is the opposite of what is observed when in cases of brain tumor part of the bulb and cerebellum may be pushed down into the spinal canal.

Dr. G. B. Hassin: I fully agree with Dr. Bassoe's opinion as to the cause of the muscular atrophy in this case. It certainly was of peripheral origin, caused by injury to the ventral roots (stretching or tearing). Situated between the spinomedullary segment and the cerebellum, the tumor originated from the inner surface of the dura and pressed on the spinal cord. Growing toward the cerebellum, it pushed the dura and stretched the roots, possibly tearing them. The posterior roots were also most likely involved, though it is difficult to explain the absence of posterior root symptoms. At any rate, the muscular atrophy was not of spinal origin as the ventral horn cells in the cervical enlargement and elsewhere showed no changes. Nor did the pyramidal tracts exhibit any degenerative changes, the clinical signs of pyramidal tract lesion in this case probably having been functional in character. We thus have here a case with a clinical picture of amyotrophic lateral sclerosis caused by an intradural tumor.

Book Reviews

DYNAMIC PSYCHOLOGY. By DOM THOMAS MOORE, PH.D., M.D. Pp. 439. Philadelphia: J. B. Lippincott Company, 1924.

"Dynamic Psychology" is a well written and worth while contribution. The author not only expresses his own conception, but also reviews the various psychanalytic beliefs and schools in the light of his own clinical experience; and, further, he subjects them to the strict measurement of actual practice. The clearness of style adds to the value of the book. The brief historical sketches are interesting and bespeak a well-tilled educational background. For instance, one reads that Aristotle in his "Parva Naturalia" wrote "Concerning Dreams" and "Concerning the Interpretation of Dreams," and had some insight into those modern psychologic disciplines which psychanalysis claims as its own. Moore defines consciousness as the "activity of a living organism of a definite type," and for him it has not wholly lost its significance because of the tremendous importance given to the unconscious. However, the latter is not only admitted but conceded to be a strong motivator of human conduct. The subconscious is an intermediate territory, the repository of those things which are neither strictly conscious nor strictly unconscious. From his study of dreams, the author can come to only a limited agreement with Freud. He is in accord with the theory that every dream contains a reference to recent experiences, but cannot accept the hypotheses relative to a universal foundation on childhood experiences, the constant presence of the sexual element, the unvarying wish fulfilment or the creation of the dream censor. He suggests, and the suggestion is not illogical, that: "In our sleeping life the sensations are largely lacking. Instead we have a train of thought and hypnagogic hallucinations. These hypnagogic hallucinations are seized upon by the train of thought, modified by it and woven into the fabric of our dreams. No censor is necessary. Dreams are symbolic because they are woven not from sensations but from hypnagogic hallucinations." The tabulation illustrates the author's classification of mental processes.

Moore differs sharply from the behaviourists. He is not able to conceive that all human conduct may be reduced to the terms of a reflex arc, no matter how complicated the arc is made by the introduction of "cortical" or "psychic" elements. Students, according to their trends, will find that the strength or the weakness of Moore's psychology is in his unwillingness to take many steps of reasoning with the assistance of the stilts of analogy. For instance, he follows Loeb in the essential mechanical nature of tropisms in plants, but doubts the sole mechanistic quality of tropisms in animals, and concludes that "the rôle played by tropisms in human life is very limited." Such caution is not unscientific. It is true that we learn by analogy, but so, too, are we sometimes deceived by its easy invitation to make extensive judgments. The chapter on the affective mental states comprises a series of self-propounded queries with an elaboration of personal reply and belief. Moore's psychology in this field may be briefly summarized. He distinguishes between mere sensory feelings and emotions on the basis that the latter are: "(1) Accompanied by a much more complex and extensive bodily resonance; (2) an emotion is a reaction to an intellectual insight and not to a mere sensation." Furthermore, the affective mental states are not attributes of sensation but independent forms of mental life, and as a corollary the affective

mental states are not sensations. Representative mental processes play a large part in the emotions, as, concretely, a slap in the face in itself does not suffice to call forth anger, but the intellectual understanding of what the slap means in the particular case is a needed component before the affect is deeply stirred. Finally, the Lange-James theory is opposed and denied. It is surprising that in the complete summary of the emotions which is presented, the author has failed to discuss the influence of physical states induced by the emotions as determining factors in prolonging the emotional status by which they were originally called into being.

Elements of mind	Mental functions	reception	attention
			perception: sensory; intellectual
		construction	association
			judgment
			reason
		conservation	sensory
			intellectual
	Mental products	representative, actions of consciousness	sensory: external—sensations; internal—images (phantasms)
			intellectual—abstract idea
		appetite reactions of consciousness	necessary: (reflex action)
			affective: feeling; emotion
			connative: impulse; desire; (instinct)
			free—act of will
	Mental dispositions	general	temperament (native)
			character (acquired, result of voluntary action)
		special habits	good—virtues
			indifferent
			bad—vices

In the chapter on "Instinct and Impulse," the argument against the behaviouristic school is continued and extended. For instance, Moore cannot bring himself to believe that the feeding of the young by the parent bird (which consists of two acts: [*a*] the regurgitation of the food; [*b*] the depositing of food in the mouth of the young) is to be viewed as reflex. It is possible that the regurgitation may be mechanically stimulated by the tapping of the fledgling; but surely the depositing of the food, which involves close motor coordination, cannot be purely mechanistic. Naturally, the premises fail utterly of application in the instance of homo sapiens, so that the author concludes that neither human instinct nor human impulse can be wholly reflex. Impulse is defined as a "tendency that we experience in the presence of actual opportunity to make use of any one of our human abilities." Desire is the "resultant of an impulse that is not or cannot be carried out"; and again, "A desire

is a craving that we experience to seek or produce a situation in which impulsive tendencies may be satisfied, or natural wants may be supplied." The principles for the management of human desires deserve to be quoted:

"1. Human life is so complicated and our abilities are so manifold and opportunities are so numerous that it is a physical impossibility for anyone to realize all his desires."

"2. All desires are not equally worth while satisfying and the criterion of worth in evaluating them is not pleasure but accomplishment."

"3. It is, therefore, necessary for us to establish a hierarchy of desires in which there shall be one supreme end of life to which everything else must conform."

"4. It is clear that in the hierarchy of our desires the lower and sensual should be subordinated to the higher and intellectual; for though the craving for the sensual may be stronger, the satisfaction that comes from the intellectual is vastly more extensive, more lasting, more productive of good to the individual and society and fraught with no evil consequences."

"5. With a clear conception of means and end in our life we must order our labors in accordance with opportunity."

"6. It is reasonable to exercise self-denial: (*a*) In order that our end may be attained. (*b*) In order that our efficiency may not be impaired."

The "Conflict in Childhood"[1] is replete with sensible advice and helpful admonition. It is a relief to note that the author has not become infected with the fallacy which is but too frequent; namely, the urge to find that tremendously complicated and deeply hidden situations are *always* at the roots of the adjustment difficulties of children. The reviewer believes with the author that not infrequently simple factors determine these situations. Some of these factors are not accessible to us, not because they are so hard to unravel, but because we have veiled our judgments with so much wisdom (?) and impedimenta that the direct and open processes of the mind of the child are no longer in the field of our vision. The elements leading to the determination of the self-ideal are: (1) accidents of the environment; (2) hereditary abilities, and to a small degree (3) organ inferiority.

Moore has coined the words "psychotaxes and parataxes." By analogy with the tropism, a positive psychotaxis is a tendency to "enjoy pleasant states of mind or to make use of pleasant emotions and feelings." The opposite tendency to avoid unpleasant situations is negative psychotaxis. The abnormal emotional adjustments, of which there are many varieties, are the parataxes. The classification which is built up is ingenious and has its valuable aspects, but new efforts at classification cannot be commended. Nosology grows slowly, and its foundation stones are accurate knowledge of etiology and pathology. It is better to use our present classification with all its flaws, and let changes wait on additions to our scientific knowledge of cause and morbidity.

The part on "Psychoanalysis and Psychotherapy" contains much shrewd comment and sound critique. The generalization of Freud and the ease with which *sometimes* was translated into *always* by him in the development of his method is scored. Certain extremities put forward by some of Freud's disciples are discounted. Moore has found that mental catharsis *alone* is inadequate as a therapeutic agent, and to shut out consideration of the patient's environmental liabilities and handicaps is to court failure. "If the present position of the patient in life is an impossible one, it can be readily understood that unburdening the past will not clear up the present. In such cases, one must not only unburden, but also find a present solution for the patient's difficulties." Finally, attention is called to the fact that the totem sacrifice, from which

Freud drew such far-reaching conclusions in "Totem and Taboo," has never been substantiated. Goldenweiser, the anthropologist, writes:

(1) "Totemic sacrifice is a phenomenon practically unknown to ethnologists."

(2) "The idea of a primitive Cyclopean family is itself a figment."

(3) "The eating of the father by the patricidal brothers is a notion which doubtless would have met with derision in the aboriginal fraternity itself. . . There has been some ceremonial eating of man, victims, as in Polynesia, of a war raid; here and there, human flesh was used in cases of severe famines. But we do not hear of the eating of relatives."

(4) "Suppose the original tragedy, the patricidal act of the brothers, had actually taken place with all the immediate psychological consequences assumed by Freud. But by what means can these facts be brought into relation with those subsequent historic phenomena of society, religion, morality, and art, the root of all of which Freud points to in that ancient enactment of the Oedipus complex in a tragic social setting?"

Nevertheless, Moore never is critical merely for the sake of being critical, and freely expresses his indebtedness to Freud for the new avenues which he opened for psychiatry. So does he utilize some of the presentations of Bleuler and Jung, and he believes the psychiatric world would have been the loser if these two students had not departed radically from the Freudian concept and system. It is a pleasure to note that the author gives a greater meed of praise to an American psychiatrist, Adolph Meyer, for his scientific acumen and constructive caution.

Considerable space is given to discussion concerning determinism vs. free will. Naturally, this question is not easy to discuss. Modern psychology gives less and less space to the consideration of the existence of the will. However, Moore has escaped the contagion of the absolutely fatalistic point of view. It is always difficult to evaluate an opinion relative to a subject of this kind, since both the trend of the author and of the reviewer must remain imponderable quantities, and in such situations, trends, attitudes, etc., are significant. Naturally, human behavior must be profoundly influenced by the particular "set" which the individual receives from all the past experiences of his life, so that choice is not without predetermined limitations. To put it concretely, let us suppose that A and B, two adults, are each confronted with situations in which the telling of an untruth will secure an advantage. The early training and environmental contacts of A all emphasized truthfulness, while those of B looked with easy complacence on the advantagious lie and even applauded its clever telling. No logic is needed to predict that A is apt to be truthful, while B is likely to fabricate. Not that Moore disputes the "set" or direction which early and even later influences give to our behavior; he simply cannot accept the concept that all free choice is excluded by them. This is not an illogical belief, and there is at least as much to be said for it as there is against it.

In conclusion, there is a chapter on the soul. Although psychology was originally the science of the soul, yet "Dynamic Psychology" is one of the few modern books which mentions it. Probably in this day, psychology, if it considers soul at all, considers it in terms of what might be covered by the term integration. Without introducing theology into the controversy, it is only fair to mention that Moore's arguments for the existence of a soul are at least as scientific as are the "proofs" of many of the qualities which are, without hesitation, credited to the fascinating but inadequately explored territory—the unconscious.